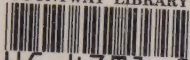


COUNTWAY LIBRARY



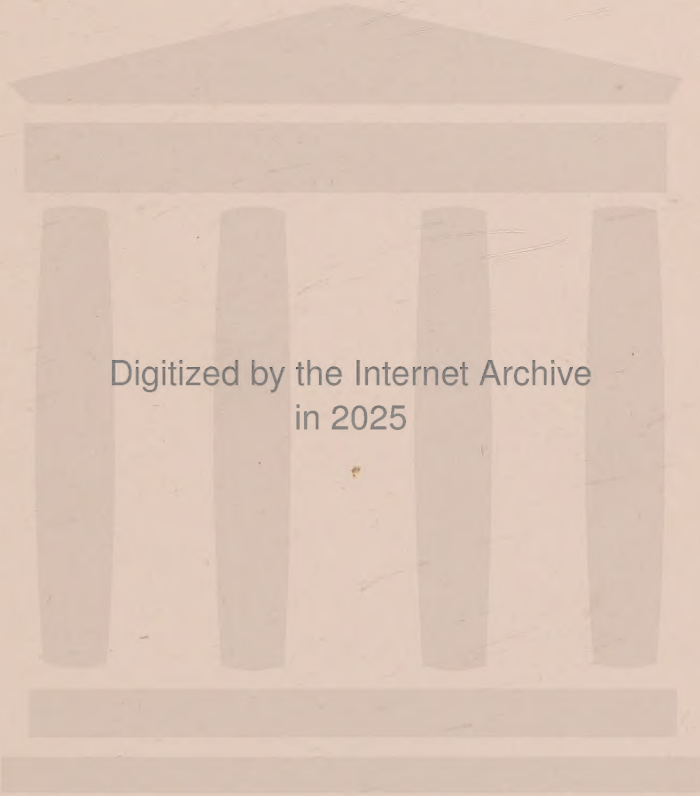
HC 4ZT1 C











Digitized by the Internet Archive  
in 2025







# HANDBOOK OF MEDICAL TREATMENT

EDITED BY  
JOHN C. DACOSTA, JR., M.D.

ASSOCIATE PROFESSOR OF MEDICINE, JEFFERSON MEDICAL COLLEGE  
PHILADELPHIA

WITH THE ACTIVE CO-OPERATION  
OF  
FOURTEEN ASSOCIATE EDITORS

IN TWO VOLUMES  
VOLUME TWO

*ILLUSTRATED*

WITH AN INTRODUCTION  
BY  
WILLIAM W. KEEN, M.D., LL.D., Hon. F.A.C.S.  
EMERITUS PROFESSOR OF SURGERY, JEFFERSON MEDICAL  
COLLEGE, PHILADELPHIA



PHILADELPHIA  
F. A. DAVIS COMPANY, PUBLISHERS

ENGLISH DEPOT  
STANLEY PHILLIPS, LONDON

1919



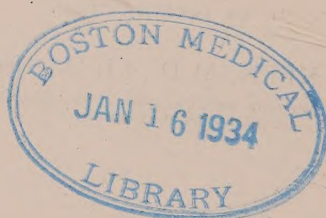
6. L. 455.

COPYRIGHT, 1919

BY

F. A. DAVIS COMPANY

Copyright, Great Britain. All Rights Reserved.



PRESS OF  
F. A. DAVIS COMPANY  
PHILADELPHIA, U. S. A.



## ASSOCIATE EDITORS.

FRANK A. CRAIG, M.D.,

Instructor in Medicine, University of Pennsylvania; Visiting Physician, Henry Phipps Institute, University of Pennsylvania; Visiting Physician, White Haven Sanatorium; Physician in Charge of the Tuberculosis Class, Presbyterian Hospital.

JUDSON DALAND, M.D.,

Professor of Clinical Medicine, Graduate School of Medicine, University of Pennsylvania.

FRANCIS X. DERCUM, A.M., M.D., PH.D.,

Professor of Nervous and Mental Diseases, Jefferson Medical College, Philadelphia; Ex-President of the American Neurological Association; Foreign Corresponding Member of the Neurological Society of Paris; Corresponding Member of the Neurological and Psychiatric Society of Vienna; Member of the Royal Medical Society of Budapest; Consulting Neurologist to the Philadelphia General Hospital, etc.

CLIFFORD B. FARR, A.M., M.D.,

Associate in Medicine, University of Pennsylvania; Assistant Visiting Physician, Philadelphia General Hospital; Professor of Diseases of the Stomach and Intestines, Philadelphia Polyclinic; Formerly Pathologist, Presbyterian Hospital, Philadelphia.

M. HOWARD FUSSELL, M.D.,

Professor of Applied Therapeutics, University of Pennsylvania; Physician to the University Hospital, the Episcopal, St. Timothy's, and St. Mary's Hospitals, Philadelphia.

VICTOR G. HEISER, M.D., D.Sc.,

Director for the East, International Health Board, Rockefeller Foundation; Formerly Director of Health for the Philippine Islands, and Professor of Hygiene and Sanitation, College of Medicine and Surgery, University of the Philippines.

J. NORMAN HENRY, M.D.,

Visiting Physician, Pennsylvania Hospital; Formerly Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; Formerly Assistant Physician, Philadelphia Hospital.

## WILMER KRUSEN, M.D., LL.D., F.A.C.S.,

Professor of Gynecology, Temple University of Philadelphia, Department of Medicine; Chief Gynecologist, Samaritan Hospital; Gynecologist, Garretson Hospital; Director, Department of Public Health and Charities, Philadelphia.

## B. B. VINCENT LYON, A.B., M.D.,

Chief of Clinic, Gastro-enterological Department, Jefferson Hospital; Pathologist, Methodist Episcopal Hospital, Philadelphia.

## HENRY K. MOHLER, M.D.,

Medical Director, Jefferson Hospital, Philadelphia.

## RALPH PEMBERTON, M.S., M.D.,

Physician to the Presbyterian Hospital, Philadelphia.

## CHARLES E. DE M. SAJOUS, MD., LL.D., Sc.D.,

Fellow of the College of Physicians and of the American Philosophical Society; Professor of Therapeutics, Temple University of Philadelphia, Department of Medicine.

## S. CALVIN SMITH, Sc.M., M.D.,

Instructor in Medicine, Jefferson Medical College; Acting Chief Clinical Assistant, Medical Clinic, Jefferson Hospital, Philadelphia.

## SAMUEL S. WOODY, M.D.,

Chief Resident Physician, Philadelphia Hospital for Contagious Diseases.



# CONTENTS

## VOLUME II.

	PAGE
DISEASES OF THE BLOOD, . . . . .	3

By JOHN C. DA COSTA, JR., M.D.

Symptomatic Secondary Anemia, 4; Chlorosis, 8; Pernicious Anemia, 17; Leukemia, 29; Chloroma, 43; Hodgkin's Disease, 44; Splenic Anemia, 47; Infantile Splenic Anemia, 50; Purpura, 52; Hemophilia, 56; Erythemia, 61.

DISEASES OF THE DUCTLESS GLANDS, . . . . .	67
--	----

By CHARLES E. DE M. SAJOUS, M.D., LL.D., SC.D., AND  
LOUIS T. DE M. SAJOUS, B.S., M.D.

Diseases of the Adrenal Glands, 67; Adrenal Insufficiency (Hypoadrenia), 72; Adrenal Overactivity (Hyperadrenia), 84; Adrenal Hematoma, 87; Hypernephroma, 88; Tumors of the Adrenal Bodies, 90. Diseases of the Thyroid Gland, 92; Thyroid Insufficiency (Hypothyroidia), 98; Infantile Myxedema (Cretinism), 103; Myxedematous Infantilism, 106; Thyroiditis, 107; Thyroid Overactivity (Hyperthyroidia) and Exophthalmic Goiter, 109; Goiter, 118. Diseases of the Parathyroids, 126; Parathyroid Insufficiency (Hypoparathyroidia), 129. Diseases of the Thymus, 133; Thymus Insufficiency (Hypothymia), 135; Hyperplasia of the Thymus, 141. Diseases of the Pituitary Body, 145; Pituitary Overactivity (Hyperpituitarism), 149. Diseases of the Pineal Gland, 168; Pineal Tumors, 171; Pineal Insufficiency and Mental Retardation, 172. Endocrinic Disorders of the Ovaries, 173; Endocrinic Ovarian Insufficiency, 177; Endocrinic Ovarian Overactivity, 182; Endocrinic Disorders of the Testicles, 184; Endocrinic Testicular Insufficiency, 186; Endocrinic Testicular Overactivity, 190.

DISEASES OF THE CARDIOVASCULAR SYSTEM, . . . . .	201
--	-----

By J. NORMAN HENRY, M.D., AND  
S. CALVIN SMITH, SC.M., M.D.

Cardiac Irregularities, 201; Pericarditis, 222; Myocarditis, 228; Endocarditis, 236; Valvular Disease, 242; Angina Pectoris, 255; Aneurysm, 259; Arteriosclerosis, 267; Blood-pressure, 273; The Use of Cardiac Drugs, 278; The Nauheim Baths, 286; Methods of Balneologic Treatment, 287; Exercises in Chronic Heart Disease, 292; Gymnastic Poses for Resistance Exercises, 298.

## DISEASES OF THE RESPIRATORY SYSTEM, . . . 325

By FRANK A. CRAIG, M.D.

Acute and Subacute Bronchitis, 325; Chronic Bronchitis, 332; Fibrinous Bronchitis, 337; Bronchiectasis, 339; Bronchial Asthma, 349; Hay-fever and Hay-asthma, 358; Emphysema, 361; Pulmonary Tuberculosis, 371; Chronic Non-tuberculous Pulmonary Infections, 472; Pulmonary Congestion, 474; Pulmonary Edema, 476; Pulmonary Abscess, 478; Pulmonary Gangrene, 484; Pneumoconiosis, 486; Pulmonary Syphilis, 489; Pulmonary Actinomycosis, 493; Pulmonary Streptothricosis, 494; Pulmonary Neoplasm, 495; Echinococcus Disease of the Lungs, 497; Pleural Fluids, 499; Acute Fibrinous Pleuritis, 502; Serofibrinous Pleuritis, 509; Purulent Pleuritis, 521; Hemorrhagic Pleuritis, 526; Pleural Neoplasms, 526; Hydrothorax, 528; Hemothorax, 528; Chylothorax, 529; Pneumothorax, 529; Bacterins in the Treatment of Diseases of the Lungs, Bronchi, and Pleura, 534.

## DISEASES OF THE KIDNEYS, . . . 541

By JUDSON DALAND, M.D., AND

FRANCIS J. DEVER, M.D.

General Considerations, 542; Displacements of the Kidneys, 553; Anomalies of Urinary Secretion, 561; Circulatory Disturbances of the Kidneys, 574; Nephritis, 578; Chronic Parenchymatous Nephritis, 589; Chronic Interstitial Nephritis, 599; Nephrolithiasis, 619; Hydronephrosis, 625; Pyogenic Infections of the Kidneys, 626; Perinephritic Abscess, 632; Tuberculosis of the Kidney, 633; Tumors of the Kidney, 639; Congenital Cystic Degeneration of the Kidneys, 639.

## DISEASES OF THE DIGESTIVE SYSTEM, . . . 643

By B. B. VINCENT LYON, A.B., M.D., AND

LOUIS LEHRFELD, A.M., M.D.

Diseases of the Mouth as Related to General Systemic Diseases, 643; Fissures of the Lips, 648; Herpes Labialis, 649; Foul Breath, 650; Gingivitis, 650; Pyorrhea Alveolaris, 651; Catarrhal Stomatitis, 655; Aphthous Stomatitis, 656; Bednar's Aphthæ, 658; Thrush, 658; Ulcerative Stomatitis, 659; Gangrenous Stomatitis, 661; Mercurial Stomatitis, 662; Carriers, 663; Epidemic Sore Throat, 665; Syphilitic Affections of the Mouth, 666; Gonorrhea of the Mouth, 667. Diseases of the Tongue: Acute Glossitis, 668; Chronic Glossitis, 669; Sublingual Ulcer, 671; Tumors of the Tongue, 671; Macroglossia, 672. Diseases of the Salivary Glands: Ptyalism, 672; Xerostoma, 672; Inflammation of the Parotid Glands, 672; Salivary Calculi, 673; Ludwig's Angina, 674. Diseases of the Esophagus: Acute Esophagitis, 674; Ulcer of the Esophagus, 675; Carcinoma of the Esophagus, 677; Esophagismus, 678; Diverticulum of the Esophagus, 679; Foreign Bodies, 681; Stricture of the Esophagus, 681. Diseases of



the Stomach and Duodenum: Ulcer of the Stomach and Duodenum, 684; Carcinoma of the Stomach, 724; Sarcoma of the Stomach, 746; Gastritis, 749; Alcoholic Gastritis, 779; Syphilis of the Stomach, 780; Tuberculosis of the Stomach, 789; Pylorospasm, 796; Pyloric Obstruction, 801; Congenital Pyloric Stenosis, 804; Cardiospasm, 810; Gastrectasis, 818; Gastric Crises of Cerebrospinal Syphilis, 826. Diseases of the Intestines: Acute Enteritis, 837; Chronic Enteritis, 839; Enteritis in Infants, 841; Cholera Infantum, 844; Acute Colitis, 845; Chronic Mucous Colitis, 845; Ulcerative Colitis, 848; Appendicitis, 849; Constipation, 854; Intestinal Obstruction, 859; Enteroptosis, Visceroptosis, Splanchnoptosis, 862; Intestinal Neuroses, 867; Carcinoma of the Intestine, 869; Tuberculosis of the Intestine, 871; Syphilis of the Intestine, 872. Diseases of the Liver: General Considerations, 874; Biliousness, 875; Jaundice, 877; Catarrhal Jaundice, 877; Acute Infectious Jaundice, 881; Acute Cholecystitis, 890; Gall-stones, 892; Chronic Cholangitis and Chronic Cholelithiasis, 894; Cirrhosis of the Liver, 898; Abscess of the Liver, 905; Acute Yellow Atrophy of the Liver, 908; Fatty Degeneration of the Liver, 909; Fatty Infiltration of the Liver, 910; Amyloid Disease of the Liver, 911; Syphilis of the Liver, 912; Tuberculosis of the Liver, 913; Tumors of the Liver, 913. Diseases of the Pancreas: General Considerations, 914; Pancreatic Hemorrhage, 916; Acute Pancreatitis, 916; Chronic Pancreatitis, 918; Pancreatic Calculi, 919; Pancreatic Cysts, 920; Carcinoma of the Pancreas, 921.

## DISEASES OF THE PERITONEUM, . . . . . 927

By WILMER KRUSEN, M.D., LL.D., F.A.C.S.

Acute General Peritonitis, 927; Chronic Peritonitis, 945; Non-exudative Chronic General Peritonitis, 952; Pelvic Peritonitis, 953; Appendicular Peritonitis, 959; Subphrenic Abscess, 960; Peritoneal Neoplasms 963; Ascites, 966.



# Diseases of the Blood.


BY

JOHN C. DA COSTA, JR., M.D.,

Associate Professor of Medicine, Jefferson Medical College, Philadelphia; Assistant Visiting Physician, Jefferson Hospital; Consulting Physician, Northwestern General Hospital; Fellow of the College of Physicians of Philadelphia; Fellow of the American Academy of Medicine; Member of the American Therapeutic Society; etc.







# Diseases of the Blood.

---

## FOREWORD.

THE progress of hematology during the past few years has added greatly to our ability to treat more intelligently those somewhat poorly defined disorders classified, more or less arbitrarily, as Diseases of the Blood.

The treatment of a given anemia should be determined by intelligent inquiry as to the exact nature of the morbid process, an investigation that at times calls for a correlation of data relating to every potential etiologic factor, to information derived from a careful physical examination, and to the suggestions furnished by the blood report. To advise iron merely because pallor happens to be the leading clinical feature of the patient is about as rational therapy as to expect to cure constipation by prescribing a laxative without search for the underlying factor of a sluggish bowel. In the treatment of an anemia, therefore, its identity must be established; the exciting factor removed, if possible; and the blood deterioration remedied by appropriate hematinics, nutritious dietary, and observance of sane hygienic rules. This routine applies in general to the treatment of all types of blood disorders—primary, secondary, and belonging to the ill-defined intermediate group. Thus, while in virtually all of these clinical entities iron and a ferruginous ration are useful, in certain of them arsenic, by the mouth or in the form of salvarsan, is indicated; while in still others nothing short of blood transfusions, radiation, or splenectomy can be relied upon, even as a palliative step.

In the following pages the reader's knowledge of modern hematology is presupposed, and laboratory technic relating thereto is omitted, but the relevant facts of the clinical pathology and diagnosis of the disease under discussion are briefly rehearsed with the intention thus to present a reasonable

basis for the therapeutic measures subsequently advised. These are presented in some detail and explicit directions given, whenever the technical methods advised justify so doing.

The choice of drugs and the method of their administration have been determined largely by the author's personal experience in various types of anemia, but current practice has not been lost sight of, and the newer special therapeutic procedures of real utility have been given attention commensurate with their value.

Hayem's dictum that "*l'avenir appartient à l'hématologie*" perhaps does not merit literal acceptance, but it is an obvious truth that hematology occupies a leading place in modern clinical medicine, and that the successful treatment of blood diseases assumes on the part of the physician an intimate appreciation of the underlying pathologic defects of such disorders, and an intelligent understanding of the various methods designed for their control. With these phases of the subject this section deals, it is to be hoped, succinctly and practically, so that the reader shall obtain helpful information, rather than theoretic discussion of moot points.

### SYMPTOMATIC SECONDARY ANEMIA.

As its name implies, this condition arises as a result of pathologic change elsewhere in the body; in this sense it is *secondary*. It is symptomatic in that the anemia is but a symptom provoked by an underlying condition, and in this respect it is to be distinguished from the *primary* anemias, such as chlorosis, pernicious anemia, splenic anemia, and the leukemias, which arise as initial conditions, the cause of which is unknown. It is that form of anemia which is most frequently encountered by the physician.

Thus, from a clinical viewpoint it is convenient, even if not accurately scientific, to group under secondary anemias those forms of blood deterioration whose origin and persistence is tangibly referable to an adequate exciting cause, the removal of which, plus the intelligent use of proper food and hematinics promptly excites regeneration of the blood along normal lines.

Such a clinical classification as the foregoing, therefore,



excludes from the secondary anemias all those types of blood deterioration which to all intents and purposes may be regarded as genuine idiopathic processes, and whose origin is based upon no discoverable cause. To this group, which are termed the primary anemias, belong chlorosis, primary pernicious anemia, and leukemia; and with these idiopathic varieties of blood diseases most clinicians also include several obscure morbid processes which, although characterized by conspicuous blood changes, do not properly come under the heading of the essential anemias. To this third and indeterminate class belong Hodgkin's disease, splenic anemia, chloroma, and infantile pseudoleukemic anemia. Finally, erythremia, purpura, and the hemorrhagic diatheses are definite blood disorders, and although they lack a consistent blood picture, are naturally classified arbitrarily in connection with diseases of the blood. It is with the foregoing types of clinical disorders that the present section deals.

Among the *causes* of symptomatic secondary anemia are such readily understood factors as a loss of blood; it may also include those dyscrasias which arise from malnutrition; those due to the absorption of metallic poisons, such as lead; those induced by toxins generated during the course of acute and chronic infections; and those referable to the hemolytic effect brought about by high fevers. Bright's disease, with its attendant defects of nutrition, produces a secondary anemia; cancer acts in a similar manner, as also does syphilis, malarial fevers, and numerous other infectious processes.

The essential feature, then, of a symptomatic secondary anemia is that it shall arise as a symptom of, and secondary to, ascertainable metabolic changes elsewhere in the economy.

The *blood picture* undergoes a variety of changes, varying in intensity with the chronicity and virulence of the underlying causative condition. Anemias due to simple defects of nutrition may show but a trifling deficiency of the hemoglobin content, with a commensurate loss of erythrocytes, and with but a moderate, if any, degree of leucocytosis. When, however, toxic factors are the underlying cause, the hemoglobin and erythrocytes are more decidedly subnormal, and the number of leucocytes is proportionately increased. In lead poisoning it is common to find a 50 per cent. loss of hemoglobin and

erythrocytes. The secondary anemias which arise in consequence of intestinal parasites cause a profound change in the blood, the picture frequently resembling pernicious anemia to such a degree that the differentiation is based solely upon the fact that the blood gradually returns to normal after the complete expulsion of the parasite.

The marked pallor of the skin and mucous membranes first attracts the attention of the physician. Following hemorrhages, the patient may complain of a faint and giddy feeling and of noises in the ears; disordered vision, labored breathing, mental confusion, rapid action of the heart, with a faint pulse, and a sense of early exhaustion are frequently added to the clinical picture.

So-called hemic murmurs, audible particularly at the base of the heart, and a venous hum over the course of the external jugular veins are additional signs of diagnostic value in many of the high-grade anemias of secondary origin.

#### TREATMENT.

The first step in the treatment of symptomatic secondary anemia is to identify and then to remove the underlying cause. If malaria be the provocative factor, quinin is indicated; if the anemia be secondary to syphilis the iodids or salvarsan are to be exhibited; if hook-worm be the cause, thymol will be administered; if cancer lies at the root of the trouble, the knife is to be employed; if an abscessed tooth, long unsuspected and continuing for years, has been productive of the hemolytic change, the care of a competent dentist is necessary before the reconstruction of the blood by dietary, pharmaceutical, and hygienic measures is undertaken.

*Dietetics.* Nutritious food, properly assimilated, is, of course, the natural blood-builder. Lost appetite may be regained by skill in the preparation of food, by the use of appetizing condiments, and by advising stomachics such as gentian, quassia, cimicifuga, and nux vomica. Meats, milk, eggs, and fats are indicated, combined in a proper proportion with starchy foods and green vegetables in a normal, rational meal. The dietary is never to be limited to any one particular variety of food, and broad, well-balanced meals are to be given the anemic subject. Veal, ham, and pork have a tendency to

upset digestion, and rich gravies, hot breads, cakes, and an excess of candy may do more harm than good.

*Drug Therapy.* Iron is the hematinic *par excellence* in the treatment of secondary anemias. It has been estimated that the body requires, for physiologic processes,  $\frac{1}{8}$  of a grain (0.00810 Gm.) of iron each day. To replace the systemic loss of this element, iron may be exhibited in the form of Bland's pill, consisting of 5 grains (0.324 Gm.) of carbonate of iron, so prepared that the drug is not permitted to oxidize or react until it reaches the stomach. (See p. 12.) Tincture of the chlorid of iron, once deservedly popular, is rarely used nowadays on account of its distinctly deleterious effect upon the teeth, it being almost impossible to administer this drug, or to swallow it, without it coming in contact with the denture. Arsenic may be combined with iron when the number of the erythrocytes is subnormal, Fowler's solution (*Liquor potassii arsenitis*) may be employed, beginning with initial doses of 3 drops after meals, increased 1 drop a day until the symptoms of physiologic tolerance appear.

Or ampules of *sodium cacodylate*, each containing 0.20 Gm. (3 grains) to the mil (16 m.) may be used in preference to the foregoing preparation of arsenic. It is the author's practice to give, by intramuscular injection, a series of twelve doses of this preparation, on alternate days, beginning with a dosage of 0.032 Gm. ( $\frac{1}{2}$  grain), and increasing by this quantity with subsequent injections until a maximum of 0.13 Gm. (2 grains) is attained.

Iron citrate, given hypodermically, is much vaunted in secondary anemias, and justly so, inasmuch as this manner of administering the metal robs its use of the many by-effects so constantly arising from taking iron by the mouth. The proper technic of the intramuscular use of iron is dealt with under the discussion of Chlorosis (page 12).

A hygienic manner of living will aid in the early restoration of the anemic subject. Fresh air, deep breathing, moderate exercise, life in the open, attention to the organs of elimination, frequent baths followed by vigorous rubbing are all part and parcel of the rational treatment of a secondary anemia of any type.



### CHLOROSIS.

In chlorosis the typical features of the blood picture show an unnaturally subnormal hemoglobin figure, with little or no loss of erythrocytes, a virtually normal leucocyte count, and a decided increase of the volume of the blood plasma which retains its normal density. The disease reflects a retardation of hemogenesis, the exact mechanism of which is conjectural, although several attractive hypotheses have been suggested upon which to build up an intelligent working knowledge of its nature. Of these the theory advanced by E. Lloyd Jones<sup>1</sup> is both reasonable and largely substantiated by clinical evidence. He suggests that a hypersecretion of the female sexual organs is the active factor of the chlorotic state, and that the blood of women thus affected in reality represents exaggerated fertility, or the accumulation of abundant material designed for fetal nutrition during the period of pregnancy.

Closely related to the foregoing hypothesis is the theory advanced by Von Jagic,<sup>2</sup> who believed that the chlorotic symptom-complex is a constitutional anomaly arising from disturbances of the internal secretions, chiefly of the genital organs, but also implicating the other ductless glands.

Bunge's hypothesis, as elaborated by Stockman,<sup>3</sup> is based upon the premises that in the chlorotic subject the presence in the bowel of an excess of sulphurated hydrogen and alkaline sulphids materially interferes with the absorption of the organic iron of the ingested food, by converting it into an insoluble inorganic ferruginous sulphid. This chemical interchange deprives the blood of its necessary ration of iron, with the result that hydrogen formation is insufficient, and hence the instrumental determination of this pigment shows abnormally low percentages, and the colored cellular elements of the blood are correspondingly pale. On the other hand, in chlorosis the administration of inorganic iron, which itself cannot be absorbed, combines with and neutralizes the sulphurated hydrogen, so that the organic food iron, thus protected, can undergo unhampered absorption and hydrogen manufacture. Bunge's theory, referred to chiefly because of its historical interest, is ingenious, but too paradoxical for cre-

dence, especially in the light of our more recent knowledge of the physical chemistry of the organic and inorganic iron compounds.

Similarly, one dismisses as inadequate the one-time popular theories attributing chlorosis to Virchow's mesoblastic hypoplasia, or an arrest of development, affecting the entire arterial system and the hemogenetic organs; to recurrent hemorrhages, particularly of the uterine and the gastric surfaces; to various disorders of the sexual apparatus; and to a form of toxic hemolysis variously attributed to intestinal and gastric decomposition.

Beyond all reasonable doubt, however, is the fact that true chlorosis is restricted to the female sex. So-called "chloritic blood" by no means indicates chlorosis, in the absence of a chlorotic symptom-complex, and this error doubtless accounts for the reported occurrence of this type of primary anemia in males; it is about as compatible as is pregnancy in men, who, despite predominantly low hemoglobin values, absolutely lack the other details of the clinical picture grouped under the disease in question.

Chlorosis, then, occurs only in females, usually those in early adolescence at or near the period of puberty, or, if met with in later life, in women whose menstrual periods habitually have been scanty or painful:

The chlorotic girl, who more often than not is a blonde, given to ready blushing, is pale, short of breath, and apparently well nourished. The pallor in the vast majority of instances is distinguished by no peculiarities other than an obviously pallid skin and blanched mucosal surfaces; exceptionally, it conforms more happily to the classical (and hackneyed) coloring of "green sickness," and in an occasional example of "chlorosis florida" or "rubra" vivid patches of color on the cheeks contrast with the alabaster pallor of the face, the whiteness of the ears and lips, and the steel blue tint of the sclerotics. The dyspnea, referable to bulbar stimulation by the insufficiently oxidized blood, is a conspicuous symptom and one that to the patient is a source of great concern. Most chlorotics retain the graceful contour of feminine adolescence and look fully nourished, despite their pallid face, and in some this appearance is exaggerated by a dropsical puffiness.

Of gastro-intestinal symptoms, constipation, chronic indigestion, with anorexia, heavy breath, and annoying pyrosis, are important; and considerable dilatation of the stomach is a common complaint. Gastric ulcer, probably due to unnatural vulnerability of the gastric mucosal lining, is a not infrequent complication which may persist stubbornly as a leading clinical feature. The appetite is most capricious, and varies from time to time, complete anorexia alternating with a ravenous desire to eat, and with a perverted craving for foods such as pickles and lemons and candy.

Functional nervous disorders are most common—tearfulness, introspection, caprice, melancholy, and, exceptionally, actual outbursts of hysteria may develop to worry the patient's family, and try her medical adviser. The familiar "hysterical bark" is attributable to chlorosis rather than to hysteria by Albutt,<sup>4</sup> who significantly refers to the cure of such coughs by iron.

Circulatory disturbances are the predominant clinical features of many chlorotics, and are present in all, to a more or less marked degree. Venous hums (*bruit de diable; venen-sausen*) generated in the jugular veins, especially the right, are audible at the base of the sternomastoid muscle, as a continuous vibratory sound, visibly intensified by the erect posture, by moderate pressure, and by inspiration. Functional murmurs at the cardiac base and at the apex are common, particularly in the pulmonic area. Enlargement of the cardiac area is practically constant, and undue arterial and venous pulsations frequently occur. Venous thrombosis is a grave, but fortunately a rare, complication.

There is a moderate enlargement of the thyroid gland in many cases, and, less commonly, the spleen is definitely enlarged; in others one sees a typical Joffroy's sign—absence of horizontal wrinkling of the forehead when the subject glances at the ceiling without moving the head.

#### TREATMENT.

The action of iron in chlorosis is an example of the specific action of a drug, for no case of this type of primary anemia fails to recover with the intelligent use of iron, and, on

the other hand, none can recover without it. Iron, then, is the basis of cure of this condition, and from the effect of this metal one can confidently anticipate prompt restoration of the blood picture to normal, and the complete relief of the symptoms, alike distressing to the patient and to the medical attendant.

As to the best form of iron to be used in chlorosis, most authorities prefer the time-honored Blaud's pill of the carbonate (*Pilula ferri carbonatis*), for administration by the mouth. This is by all odds the most satisfactory form of iron to be employed for the cure of chlorosis, inasmuch as its astringent properties are mild and its long-continued use does not tend to produce unpleasant effects such as headache, indigestion, and constipation. Should, however, the bowels become sluggish after the prolonged use of this form of iron the pill may be combined with moderate doses of cascara sagrada or with phenolphthalein, or, if thought advisable, the bowels may be kept free by the use of a good sour milk, as advised by certain Continental authorities; or baker's yeast may be used to counteract this tendency, and for this purpose half a cake of yeast dissolved in a tumblerful of cold water should be taken twice daily.

Excessive ferruginization is no longer regarded as good practice, but unfortunately, where therapeutic tradition rules, this mistake is still practised to some extent. The average patient can take, without disconcerting by-effects, approximately three or four Blaud's pills daily for the first week, six pills daily for the second, and from nine to twelve daily for the third week, and thereafter until the blood picture returns to normal. Discontinuance of the iron should be gradual, at the rate inverse to that governing its progressive increase during the initial few weeks of treatment. According to the plan just outlined,  $7\frac{1}{2}$  grains ( $\frac{1}{2}$  Gm.) of iron are given daily during the first week, 15 grains (1 Gm.) daily during the second week, and about  $22\frac{1}{2}$  grains (1.5 Gm.) daily for the third week, and thereafter until a cure is effected. This amount of iron is usually well tolerated, without the fear of any discomfort to the patient; it is needless to add that this dosage is more than amply sufficient for the purposes sought—to stimulate hematopoiesis, to store up an excess of iron in



the body for subsequent absorption, and to preserve chemically the food iron in the intestinal canal.

The ordinary Blaud's pill as purchased in the drug store may be absolutely inert, and fail utterly to meet the essential conditions just named in the preceding paragraph. It may represent but an irritant oxid, in view of which it is best, if one is not sure of the exact effect of the average "stock" pill, that the metal should be prepared freshly by the druggist in small quantities. An exceptionally active Blaud's pill, very popular in Great Britain, is so compounded that no interaction of its ingredients occurs until it is acted upon by the gastric juice, as a result of which action a nascent carbonate is produced in a readily absorbable form,

If for any reason Blaud's pill should be contraindicated in the individual case, the citrate of iron may be used instead, by intramuscular injection. This of course obviates all the possible ill effects of oral administration, furnishes an immediate source of iron to the chlorotic blood, and obviates all effects of this metal upon the teeth, the stomach, and the gut. Ampules containing 3 grains (0.195 Gm.) of citrate of iron are on the market; used in connection with a No. 3 hypodermic needle and a Leur type syringe, injections deep into the deltoid muscle are made without pain or other discomfort to the patient. As a rule, it is sufficient to give these injections on alternate days.

Some authorities prefer to substitute for Blaud's pill sulphate of iron in 1-grain (0.065 Gm.) pills during the first week of treatment, this initial dosage to be doubled and trebled as the treatment progresses. With this form of iron, however, gastric disturbances and constipation are much more likely to occur than with the two forms of iron just described.

In patients with habitual loss of appetite, it may be thought better to choose the alcoholic liquid iron as represented by the tincture of the chlorid.

There is no excuse whatever for resorting to the expensive proprietary organic irons, for none of them gives more adequate results than the forms just mentioned, and with none is the hemoglobin increase more rapid and more stable than it is when one of the standard iron salts is used.

In addition to iron, arsenic is indicated in many chlorotic

girls to counteract the subnormal number of erythrocytes. Of course the demand is not great, inasmuch as the count of cells is but moderately subnormal, but in order to start the upward wave toward an excess of blood constituents, it is wise to use some form of arsenic, such as Fowler's solution or arsenous acid in order to accomplish this end. Thus the use of from 2 to 10 drops (0.133 to 0.666 mil) of *Liquor potassii arsenitis* thrice daily exerts an active stimulus of the bone marrow, excites adequate secretion of the thyroid gland, and provokes neither gastric nor renal irritation, except in highly susceptible subjects; arsenous acids, if chosen, should be given three times a day in doses of  $\frac{1}{30}$  grain (0.002 Gm.).

Although iron is the specific for chlorosis, other therapeutic measures must receive due consideration so as to deal adequately with the peculiar symptom-complex. The special symptoms relating to the chlorotic condition include a long medley of complaints, both real and imaginary, and the correction of these symptoms, individually of no great moment save to the patient herself, is a detail of treatment which should not be dispensed with in the face of favorably progressing blood reports.

Rest in bed is just as essential as the use of iron, and by this is meant a literal rest, without any physical exertion whatever, and with an environment such as to banish care, worry and all contributing factors of a disturbing character. Particularly is rest desirable in chlorotics with prominent cardiovascular symptoms, in whom complete physical relaxation, aside from its other good effects, may entirely eliminate distressing complaints, such as dyspnea, vertigo, cardiac palpitation, and edema. After, say, a fortnight or three weeks of absolute confinement to bed, and after decided improvement of the subjective symptoms has occurred, the patient may be advised to spend her mornings in bed, and, if weather conditions permit, to pass the greater part of the afternoon outdoors in the sunshine. Active physical exercise is a great mistake, for the chlorotic's languor and asthenia, despite her apparent good nourishment, are real, and readily aggravated by physical exertion.

In conjunction with the foregoing measures, systematic hydrotherapy and massage are most useful. In the patient's

own home this régime may consist simply of a twenty-minute hot 104° F. (40.0° C.) tub bath, followed by a cool or cold douche, a brisk rub, and an hour's rest.

As to the dietary, one should aim to provide a high content of proteins and fats. Unfortunately, the average case of chlorosis shows a pronounced aversion to the very source of food demanded by her thin blood. Moreover, the digestive disorders, so commonly the conspicuous symptoms of the case, further interfere with the institution of a dietary demanded by the underlying condition. The use of the various digestive enzymes, and, in extreme cases, of lavage, tend to counteract these defects. As to the most useful kind of diet to be prescribed in chlorosis, a full ration of butter, clotted Devonshire cream, rich raw milk, and eggs should be the mainstay of the dietary. If raw milk disagrees with the patient, lactic acid milk prepared with pure Bulgarian cultures, or peptonized milk are useful substitutes. Overfeeding with milk and eggs should be carefully avoided, and the exact quantity for each day's feeding is to be determined in the individual case, with due regard to personal adaptability. In addition to butter, the use of fat in the form of crisp fried bacon is generally well relished and digested; only exceptionally must one give fats in such a distasteful form as codliver oil or olive oil.

The free use of beef, served rare, should supplement the measures already indicated. The average patient can be induced to eat about 6 ounces (169.8 Gm.) of meat in each twenty-four hours. Beef juice and the concentrated beef broths, fresh fruit, together with watercress, lettuce, spinach, asparagus, and other greens help to vary the monotony of the beef diet, but starchy vegetables, such as potatoes, beets, peas, and corn should be used sparingly. As an early morning potion designed to rid the gastric mucosa of irritant mucus and to free the bowels, any of the alkaline laxative waters may be used—effervescent sodium phosphate, Rubinat, Carlsbad Sprudel, and Hunyadi. Alcohol is not indicated, although it is frequently the custom to allow a glass of claret, Burgundy, or stout to be sipped with luncheon or dinner. The peculiar craving for acids, so commonly a complaint of the chlorotic girl, may be effectually met by the use of Ringer's effervescent lemonade, prepared by adding to half a pint (240 mls) of iced

water the juice of one large lemon, sweetening with a lump or two of sugar, and made effervescent by the addition of half a teaspoonful (1.9 mls) of sodium bicarbonate.

As intimated above, there are many imaginary complaints to be dealt with. In the average case of chlorosis symptoms that demand advice rather than drugs, are the source of great distress to the patient. In such instances, counsel, a cheery outlook, encouragement about fancied ills, go a long way to better matters, and the use of any depressant or hypnotic drugs is distinctly bad practice.

Apart from these functional complaints, there are certain symptom-groups in every case of chlorosis which demand accurate and radical therapeutic measures. One must be on one's guard accurately to recognize these real complaints, and from time to time intelligently to relieve such symptoms, which in brief may be referred to the gastro-intestinal tract, to the cardiovascular apparatus, and to the nervous system.

Of the *gastro-intestinal symptoms*, constipation, coated tongue, flatulent dyspepsia, and a heavy breath are the familiar ills complained of; peptic ulcer complicates a small percentage of cases; in others the stomach is abnormally low and dilated, and in nearly all there is decided impairment of appetite, if not a complete anorexia. It is best to obviate constipation by urging the free use of a dietary consisting largely of green vegetables, fruits, salads, and fats. When such methods require to be supplemented, enemata are indicated, and if this also fails to relieve the constipation, small doses of cascara sagrada, phenolphthalein, and aloin are useful. Whatever means employed, the stools should be kept soft and the formation of scybilla prevented. If hepatic inaction and inspissation of bile exists, a course of acidum sodium oleate and sodium salicylate usually will cause a free passage of bile. For the flatulent dyspepsia and its attendant symptoms, a long list of so-called antifermentative and antiseptic medicaments is available. The following will be found to be quite as useful as any and may be given for an indefinite period with good effects: Taka-diastase, pancreatin, salol, and extract cascara sagrada, each 3 grains (0.195 Gm.) to be given at mealtime. For a gastric indigestion it is wise to use a solution, each dram of which contains 3 grains (0.195 Gm.) of



scale pepsin and 5 drops (0.30 mil) of dilute hydrochloric acid, this to be given in 2-dram (8 mils) doses, well diluted, before meals; this combination may be compounded with orange flower water and glycerin to make an agreeable mixture.

Pick's contention holds that chlorosis is excited by a hemolysis, due to toxin generated within and absorbed from a dilated stomach. However this may be, the presence of gastrectasis demands attention in a considerable proportion of chlorotics, and this complication virtually demands as much attention as the underlying blood defect. A gastrectatic dietary is indicated, with perhaps the use of the stomach tube and the administration of some of the antifermentatives just referred to. The dietary also requires most careful supervision in such cases, and on this point it will be sufficient to note here that concentrated "dry" foods of small bulk are to be chosen rather than foods which overload the stomach and tend to remain within the stomach for a protracted period.

In cases of complicated gastric ulcer Leube's régime or any one of the accepted plans of routine treatment for this condition should be adopted, and such drugs as bismuth, silver nitrate, and opium be intelligently prescribed. (See Gastric Ulcer.)

Of the many nervous symptoms inseparable from chlorosis, conditions of apathy, mental depression, melancholia, and hysteria are the more serious, while fretfulness, unjust criticism, petulance, and ill temper, demand just as careful attention. These symptoms require not so much drugs and nervines as they do firmness and suggestive measures on the part of the physician. When drugs are indispensable, strontium bromid and the isovalerianates are to be relied upon rather than the average hypnotic. Opiates are never to be used. When the nervous symptoms consist largely of tri-facial neuralgia and various neuralgic pains elsewhere, one of the less depressant coal-tar analgesics may be employed, and for this purpose acetphenetidin combined with camphor monobromate and salol are to be employed. The nervous cough so common in chlorosis needs no special treatment, but its presence should prompt the medical attendant to identify it merely as a nervous bark and not as the result of an incipient phthisis. •

Cardiovascular symptoms which are purely inorganic in character will disappear as the patient's blood improves. Meanwhile, they are to be controlled by rest and the ice-bag rather than by the use of any of the so-called cardiac drugs.

### PERNICIOUS ANEMIA.

Pernicious anemia represents a form of fatal blood deterioration depending upon a reversion on the part of the bone-marrow to an embryonic type of hemogenesis and characterized by the presence in the circulating blood stream of numerous nucleated erythrocytes, conforming histologically to those incident to fetal life (megaloblasts, mesoblasts). Associated with the foregoing distinctive retrograde change, there is an extreme degree of hemoglobin loss and even a more pronounced decline in the number of circulating erythrocytes—a peculiarity indicating a disproportionately high content of hemoglobin in the individual red corpuscles, and referred to by hematologists as a condition of “high color index.” Hand in hand with these changes, which grow progressively more striking as the disease advances, microchemical alterations and stroma degenerations appear in many of the erythrocytes. These cells lose their normal biconcave, disc-like contour, and even coloring, and undergo curious and bizarre distortions of size and shape (poikilocytosis; schistocytosis; microcytosis; megalocytosis); develop basophilic granulations where normally the stroma stains faintly acid (granular basophilia); and show diffuse and splotchy patches of basic degeneration in the cell protoplasm (polychromatophilia; polychromasia).

The leucocytes undergo no conspicuous change, although their number is generally diminished, and, differentially, it is found that a relative lymphocytosis exists, at the expense of the polymorphonuclear neutrophiles, whose number is correspondingly diminished. Small, dwarf myelocytes commonly occur in small percentages, and the proportion of eosinophile leucocytes is subnormal.

In the average well advanced example of pernicious anemia a blood examination which shows a hemoglobin percentage below 20, an erythrocyte count in the neighborhood of 1,000,-

000 cells to the cubic centimeter, and a leucocyte count of approximately 1000 represents the degree of blood impoverishment ordinarily encountered.<sup>5</sup>

The changes in the bone-marrow responsible for the blood picture reflect an effort on the part of this organ to compensate the coexisting blood destruction. They consist, in brief, of a softening and hemorrhagic condition regarded, not as a primary causal change, but as a secondary lesion, the direct result of the inroads of toxemia and hemolysis. The marrow loses its normal yellowish color, becomes red, and contains large numbers of nucleated erythrocytes similar to those found in the circulating blood, together with numerous myelogenous cells so hyaline and so delicate as almost to escape detection.

Aside from the foregoing alterations in the bone-marrow, an individual dead of pernicious anemia shows widespread evidences of a fatal hemolytic anemia, in the extensive splenic and hepatic pigmentation, and in the excess of urinary and fecal blood-pigment found at autopsy. The pigmentation of the liver, made up of highly ferruginous material, affects particularly the cells at the lobular peripheries, and the lymphatic endothelial cells and capillaries. Similar proofs of hemolysis are found in the pigmented and enlarged spleen, in which viscus the pigment granules occur both in the vascular walls and are disseminated, intracellularly and free, throughout the structure of the organ.

Eppinger<sup>6</sup> believes that anatomic changes in the spleen in pernicious anemia largely accounts for the excessive erythrocytic destruction which takes place in this organ, the exact site of the hemolysis being the splenic pulp which the erythrocytes readily permeate owing to vascular defects peculiar to this form of anemia, and here are destroyed in large numbers. This author describes in Addisonian anemia under the term "blood lymph-nodes" certain structures histologically similar to, and presumably functioning like, the splenic tissue. In subjects not benefited by removal of the spleen (*q. v. i.*) he attributes the failure to the fact that these nodes carry on the hemolytic action originally exhibited by the spleen before operation. Other evidence of the underlying hemolytic factors of this type of anemia is seen in the undue

amount of urobilin found in the stools, and as shown by Sel-lards and Minot,<sup>7</sup> in the readiness with which hemoglobinuria follows the subcutaneous injection of solutions of hemoglobin in quantities tolerated by other forms of anemia without the appearance of this blood pigment in the urine.

Secondary changes affect the cardiovascular system, giving rise in some instances to spontaneous hemorrhages and to extreme cardiac debility; and the liver and kidneys are prone to suffer similar alterations.

The entire alimentary tract is commonly affected, oral sepsis, septic gastritis, and septic enteritis being the three most frequent lesions, to the effects of which W. Hunter<sup>8</sup> is inclined to attribute the disease under discussion, believing that the excessive hemolysis in the portal area is directly due to the action of an unknown specific toxin elaborated in the gastro-intestinal canal. Squier<sup>62</sup> suggests that the hemolysis may be referable to the action of either undigested proteins or putréfactive poisons which reach the blood stream through a breach in the mucosa of the gastro-intestinal tract. In the absence of definite proof, the specific action of this unidentified toxin in pernicious anemia can be accepted only upon hypothetical grounds, although its predisposing action is easy to credit.

In a subject constitutionally susceptible to the action of blood-dissolving agencies, the existence of a profound disturbance of lipid metabolism resulting in diminution of the antihemolytic properties of the whole blood may prepare the way for the direct action of the hemolytic substance, especially if this defect be associated with disordered functions of the ductless glands. In this speculation as to the identity of the toxin responsible for Addisonian anemia, the action of hemolytic toxins in other grave anemias naturally is brought to mind, and one appreciates a comparable condition in the hemolytic anemias referable to oleic acid in *Bothriocephalus* infection,<sup>9</sup> to the oxyphenylethylamin poison derived from the *Bacillus coli communis*,<sup>10</sup> to absorption through a pathologic intestinal mucosa of foreign protein,<sup>11</sup> to the poisonous action of oestrin,<sup>12</sup> and to the placental hemolytic substance active in pregnant women.

The spinal cord changes are, as a rule, quite conspicuous,



and consist of more or less marked sclerosis of the posterior, lateral, and anterior columns, with commonly very minute hemorrhagic foci distributed through the substance of the cord. Less commonly, degeneration of various peripheral nerve fibers are found, as a consequence of neuritis.

Here may be mentioned a rare type of fatal anemia, known as *aplastic anemia*, generally regarded as a form of pernicious anemia in which virtually no response of the bone-marrow to hemogenesis exists. Such cases run a rapidly fatal course, characterized by progressive and extreme hemoglobin and erythrocyte losses, a low color index, a very scanty proportion of erythroblasts, or none at all, and a low leucocyte count, with a high lymphocyte percentage, and an absence of myelocytes. The marrow in aplastic anemia is in a state of hypoplasia, or indeed it may be actually aplastic; contains few, if any, marrow cells; and shows either a yellowish tint or is quite colorless. The obvious contrasting features of the aplastic variety and anemia of the Addisonian type relate chiefly to the pathology of the bone-marrow and to the differences in the blood pictures—common in picturing an extreme anemia, but differing essentially by having a low color index and absence of megaloblasts in the former, and a high color index and a predominance of megaloblasts in the latter.

### TREATMENT.

Intelligent management of a patient affected with pernicious anemia includes the treatment of the inherent and progressive blood deterioration, and the care of the distressing complications of the underlying hemolytic process. To the first factor are referable symptoms such as dyspnea, vertigo, syncope, languor, undue prostration, and cardiac disturbances. Physical signs like mucosal blanching, pallor and lemon-yellow tingeing of the skin, scattered small subcutaneous hemorrhages, edema of the ankles, enlargement of the liver and spleen, muscular flabbiness combined with apparent preservation of body fat, and a soft, full pulse with tremulous throbbing of the superficial vessels complete the clinical picture.

In numerous instances these leading clinical features are persistent, and intractable indigestion, obstinate and enfeebl-

ling attacks of diarrhea, nausea, and vomiting cause concern and call for urgent treatment. The mouth, often the site of disgusting pyorrhea, also must be carefully looked after, and other focal infections eradicated. In this endeavor, a thorough search is to be made for stomatitis, adenoid, tonsillar, and dental infections; and septic lesions of the stomach, appendix, gall-bladder, kidneys, urinary bladder, and uterus. Surgical treatment for such potential factors of the anemia must be decided upon so soon as the patient's hemoglobin approximates 80 per cent. of the normal standard, and sooner, if the blood shows no definite improvement, apparently because of the activity of the area of focal septic absorption.

Absolute rest in bed is essential for the satisfactory treatment of pernicious anemia; and when possible, a private room in the hospital with a special nurse, to administer to the patient's most trifling needs, should be engaged, in order to assure the best results from the therapeutic measures employed. This is insisted upon by Barker,<sup>63</sup> who also advises a strict milk diet, especially during the first seven days of confinement to bed, for which period the patient receives every two hours from seven A.M. to nine P.M.  $2\frac{1}{2}$  ounces (75 mls) of milk on the first day, the two-hourly amount being increased each day until by the sixth day 3 quarts (liters) are given in each twenty-four hours. After the first week, the strict milk diet is substituted by a ration rich in protein, fat, and carbohydrate, and the patient is urged to take, in addition to three full meals a day, several raw eggs, a quart and a half ( $1\frac{1}{2}$  liters) of milk and one-half pint (236 mls) of cream each day.

*Arsenic*, for almost half a century depended upon as the most powerful single agency available in the treatment of pernicious anemia, has not been replaced by any more efficacious drug up to the present writing.

In the last few years, salvarsan and neosalvarsan, containing respectively 31.6 and 21.1 per cent. of metallic arsenic, have been used to some extent as a substitute for Fowler's solution, on the whole with results that promise their continued use as the most certain form of therapy in this disease.

Salvarsan may be given intravenously, exactly as it is administered in syphilis, but in smaller doses and at less frequent intervals. Boggs<sup>13</sup> advises a dose of 0.3 grams (4.6 gr.)

once every four weeks, the injections being repeated until the blood picture reaches an approximately normal figure.

Neosalvarsan is preferred to the stronger salt by Byron Bramwell, who introduced the arsenical treatment of pernicious anemia in 1875, on the premise that if this metal is beneficial in fatty heart, it also might be of use in this grave form of anemia, which so constantly is associated with such a condition. Neosalvarsan is administered most satisfactorily by intramuscular injections of from 0.3 to 0.6 grams (4.6 to 9.2 gr.), repeated at the same intervals adopted in giving salvarsan, and continued until similar effects are apparent. Of the two drugs, salvarsan is regarded as perhaps the more hazardous, although decidedly more rapid, certain, and permanent in its effects, and apparently is peculiarly indicated in pernicious anemias, with coincident syphilis, and in those with a pronounced arsenic idiosyncrasy. In favor of neosalvarsan are its adaptability to subcutaneous injection without marked local inflammation; its relatively mild systemic reaction—fever, tachycardia, vomiting, anaphylactic shock—and the sustained and continued action exerted owing to its slow absorption. A point in favor of both salvarsan and neosalvarsan is their slight tendency to give rise to peripheral neuritis and other untoward symptoms of arsenical intolerance, which so commonly bar the continued use of Fowler's solution in progressing dosage.

On the whole, the use of salvarsan and neosalvarsan marks a distinct advance over the older arsenical preparations in the treatment of Addisonian anemias, and bids fair wholly to supplant the routine employment of *Liquor potassii arsenitis*, arsenous acid, and sodium cacodylate. Bramwell records<sup>14</sup> 33.3 per cent. of "temporary complete recoveries" under salvarsan, in contrast to 12.7 per cent. under Fowler's solution; the number of cases treated being 110 in the first group, and 21 in the second. As to the permanence of "cures," no definite conclusion is warranted until the treatment has endured a more convincing test of time.

Preparations of arsenic other than salvarsan still enjoy considerable vogue in certain quarters, and merit at least parenthetical mention, in case the use of the newer drug be contraindicated for any reason. Salvarsan should never be

given in the face of renal irritation, and should be used guardedly, in small doses, in subjects of cardiac weakness, striking asthenia, gastric irritability, and tendency to anaphylactic shock.

If circumstances bar the use of salvarsan or neosalvarsan, one must be content to resort to one of the older arsenic preparations of which Fowler's solution is, on the whole, the best tolerated and most efficacious in the great majority of those who are forced to undergo the protracted medicinal use of this metal. The exact point of tolerance to arsenic varies within wide limits in the individual case; some are taken with abdominal pain and diarrhea after several day's administration of but a few drops of Fowler's solution, while others may take without discomfort relatively large doses for weeks at a time.

In dealing with the average patient, it is my accustomed routine to administer a maximum daily dose of approximately 30 minims (2 mils) of Fowler's solution in three or four equal doses, well diluted, and taken after food. The initial dose is ordinarily fixed at 5 minims (0.30 mil), and the maximum amount is reached by gradually increasing the dose by a single minim (0.06 mil), say, every second day. Thus the fullest possible effect of the drug may be exhibited, usually with no risk of the gastric and renal irritation so prone to be excited when arsenic is used with a freer hand from the beginning. Only in the more fulminant cases of pernicious anemia (and these are best treated by some other methods) should one risk the by-effects of a more vigorous *régime* with the drug in question. In acute exacerbations of Addisonian anemia, Chauffard's method of administering Fowler's solution has a definite place in therapy. This author, in company with Laederich, uses a 1 per cent. aqueous solution of the drug with 1.33 per cent. sodium chlorid, of which a maximal dosage of 20 minims (1.25 mil) are given hypodermically each day, for ten consecutive days, each of these periods of treatment being separated by a week's rest. This routine has been followed by excellent results, and is reported to be unproductive of arsenical intolerance on the part of the patient.

Voga and McCurdy<sup>15</sup> are strongly in favor of *blood transfusion* as a promising means of producing satisfactory remissions of considerable duration. Undertaken at an early stage



of the disease, the transfusion of physiologically unaltered blood is regarded by these authors as a palliative treatment of great promise. This provided that the technic assures the use of blood proved to be mutually congenial to donor and recipient and that small quantities of blood are introduced at intervals determined chiefly by repeated examinations of the patient's blood with a view to estimating from time to time the regenerative reaction of the bone-marrow.

The *intramuscular injection* of defibrinated blood has numerous advocates on the Continent, and recent reports credit the procedure with freedom from systemic ill-effects, and with rapid and satisfactory improvement of the blood picture and of the patient's general health. The quantity of blood injected varies from 20 mils (5.4 f3) used as an original minimum dose, to 70 mils (18.9 f3) advised as the maximum quantity by Zubryzcki.<sup>16</sup> Ordinarily the number of injections required to bring about substantial improvement is from three to five, given at intervals of about five to seven days.

The injection by the intravenous route of not more than 5 mils (1.35 f3) of defibrinated blood is advised by Weber<sup>17</sup> as a method of treating pernicious anemia followed by results comparable to those obtained with arsenic. He cautions against using a larger amount of blood than the quantity specified, owing to the likelihood of provoking dangerous reactions and because of the better therapeutic effect of small doses.

The technic followed by Archibald,<sup>64</sup> of the Mayo Clinic, calls for from one to four transfusions usually of 500 mils (16.9 f3) of the donor's blood, although in some instances good effects were noted with much smaller quantities—50 to 100 mils (1.69 to 3.38 f3). In this series of 25 cases thus treated 69 per cent. were immediately benefited, and a direct relationship was traced between the procedure and the subsequent remission of the symptoms. Chronic cases, with a history of remissions, appeared most likely to be favorably affected, and those unaffected by the first transfusion frequently responded when a different donor was chosen for later operations.

*Venesection* has been advised, merely as a palliative measure in cases resistant to the usual methods of treatment. The temporary benefit thus established doubtless depends partly

upon a stimulation of the hemopoietic tissues and to some extent upon the withdrawal of the blood toxins incident to the disease. The technic does not differ from that of ordinary venesection, the blood being taken from a vein at the elbow. From 50 to 150 mils (1.69 f $\bar{5}$  to 5.07 f $\bar{5}$ ) are withdrawn at each *séance*, to be repeated at intervals of a fortnight during a period of about three months. In most instances the dual benefit of the operation is shown by appreciable improvement of the blood picture and amelioration of the evidences of toxemia.

The treatment of pernicious anemia with various *serums*, notably antidiphtheria, antistreptococcus, and antistaphylococcus, has been undertaken on the basis of their beneficial effect in the high-grade secondary anemias, and on such a premise organotherapy also has been employed, singly and combined. Thus Bartolotti<sup>18</sup> advises in cases refractory to arsenic and iron therapy, the use of 1000-unit injections of antidiphtheria serum, repeated three or four times at intervals varying from fourteen to forty days, in combination with extract of spleen and spinal cord, gradually increased from 0.5 gram (8 gr.) to 2.75 grams (38½ gr.) per diem. Under this routine one of Bartolotti's patients showed normal hemoglobin and erythrocyte figures and differential leucocyte count six months after suspension of the treatment. In this connection it is of interest to note the reputed cures of a number of cases of pernicious anemia reported by Mikhailoff,<sup>19</sup> who resorted to the hypodermic use of splenic extract alone, in doses of 2.5 mils (40 m.) of a 2 per cent. solution. In one patient to whom fifteen such treatments were given, six months after their discontinuance the blood was normal in every detail.

The use of antidiphtheria serum, of obvious utility in combating other types of toxic anemias, has a certain empiric place in the therapeusis of Addisonian anemias, at least in those examples of the disease in which the toxemia predominates and counteracts the attempts to better the blood deterioration by arsenical treatment. The same comment appears to be justifiable with regard to the use of splenic extract in a similar group of cases. As a radical cure, however, no reports are available to warrant such a hope from either serum treatment or organotherapy.

Of recent years *splenectomy* has become recognized as a justifiable surgical procedure in selected cases of pernicious anemia, although the operation should be undertaken merely as a means of inducing a remission usually of more striking character and of longer duration than the abatement of the active symptoms so frequently met with as a spontaneous change or as one due to active arsenization of the subject. On the basis that removal of the spleen is often followed by a conspicuous hemoglobin and erythrocyte increase<sup>20</sup> by signs of hyperactivity of the bone-marrow,<sup>21</sup> and by laboratory findings indicating diminished hemolysis,<sup>22</sup> it would seem rational to interfere surgically as a curative measure in this disease. But unfortunately splenectomy accomplishes nothing more tangible than temporary improvement. In numerous instances the tenure of life after the operation has been measured by years, with a general amelioration of the most distressing phases of the illness. In the recent study by Krumbhaar<sup>23</sup> of the exact utility of the operation, the details relating to 153 cases are analyzed. Of 27 cases at the end of the first year after operation, 9 were dead, 7 had relapsed, and 11 had improved; after two years, the figures read one, two, and three, respectively for the 6 patients still under observation, but no radical alteration of the original blood picture of pernicious anemia had been encountered. The immediate post-operative mortality of splenectomy in subjects of pernicious anemia is approximately 20 per cent., and of those who survive the operation about 65 per cent. undergo real improvement in the blood picture and in the general clinical features of the affection, no appreciable improvement occurring in 16 per cent. of the second group of cases. The post-operative improvement was generally of transient nature, and likely to be interrupted by death from intercurrent infection or a fulminant type of relapse.

Splenectomy is absolutely contraindicated in the aplastic forms of pernicious anemia, and it is rarely helpful in cases in which conspicuous and progressive blood deterioration predominates, and in those with active symptoms of spinal sclerosis. The procedure is of service in middle-aged or young subjects whose blood shows regenerative signs (erythrocytic reticulation, erythroblastic crises, Howell-Jolly bodies, in-

crease of platelets), and is not strikingly below the quantitative and qualitative normal standard, and whose clinical picture, aside from the diagnostic blood-changes, is characterized by a reasonable degree of hemolysis and by moderate splenic enlargement. A post-operative laboratory finding of real worth is the diminished urobilin content of the stools in splenectomized patients to which evidence of lessened hemolysis Eppinger<sup>24</sup> attaches great significance.

When combined with other methods of therapy, such as full arsenization, *radiation* is to be regarded as helpful in selected examples of Addisonian anemia, but neither in combination—much less alone—can it be relied upon to accomplish a radical cure for this affection.

Exposure to the Röntgen ray improves some cases of pernicious anemia, but quite fails to influence others, and no real curative influence can be attributed to this form of treatment. It is particularly those instances in which the bone-marrow degeneration has not progressed to a point where this tissue fails to respond to stimulation in which radiation is helpful, and in such cases the improvement may be prompt and striking, although unfortunately, not lasting in those treated with  $x$ -ray alone, unsupported by other measures.

The same technic as that used in leukemia (*q.v.*) is satisfactory in pernicious anemia, and the progress of the treatment should be followed by the results of repeated blood examinations, which show an increase in the number of erythroblasts in patients undergoing improvement, but no such evidence of blood regeneration in those whose marrow is uninfluenced by the rays.

Of the various *special symptoms* which may demand attention in the subject of pernicious anemia, the gastro-intestinal disturbances are by all odds the most common and troublesome to manage. Acute gastro-enteric manifestations instinctively call to the physician's mind arsenic intolerance as the exciting cause, and with equal meaning the subject of the patient's dietary. The simple test of withholding arsenic for a time, together with a strict revision of the diet, will settle the question as to whether the disturbances were due to such factors as these. If this does not better matters, a more definite method is to be adopted. In this connection it



must be recalled that the care of the mouth is to receive attention; dental caries is to be treated; and ulcerated gums must be healed before one can believe that all factors of gastrointestinal symptoms are eliminated.

Many cases of pernicious anemia are subject to capillary hemorrhages, and this sort of bleeding usually can be controlled readily by the various hemostatic drugs. Oozing from the mouth or nose is best treated by the topical application of adrenalin chlorid in 1:1000 solution, of fluidextract of hamamelis, or of glycerite of alum; the use also of coagulin (Ciba.) is applicable in this type of bleeding. Packing the nares with sterile gauze strips, soaked with 1:2000 adrenalin chlorid solution, will usually immediately control a troublesome nose-bleed. If there be retinal hemorrhage, it is advisable to use one of the iodids, preferably sodium iodid, in doses of from 15 to 30 grains (1 to 2 Gm.) daily to favor absorption of the clot.

Intestinal bleeding calls for temporary withdrawal of all food by the mouth. Iced compresses to the abdomen, the insistence of rest in bed, and the use of appropriate styptics, which act upon the mucous surface of the bowel are useful in this emergency. A favorite pill for this purpose is composed of 1 grain (0.065 Gm.) lead acetate, 2 grains (0.130 Gm.) of camphor, and  $\frac{1}{4}$  grain (0.016 Gm.) of opium. Monsel's solution of subsulphate of iron is another drug much used in intestinal bleeding; it should be administered in 3-grain (0.195 Gm.) salol-coated pills, to insure its reaching the intestinal canal undissolved. In order to control a hemorrhage in the lower bowel the following rectal injection will prove useful: enemas of iced water; of 2 per cent. alum solution; of 2 per cent. tannic acid; of 5 per cent. Monsel's solution; of  $\frac{1}{4}$  per cent. silver nitrate; of  $\frac{1}{2}$  per cent. argyrol. Hematemesis, which is a most unusual symptom, does occasionally occur, and is a source of great alarm to the patient, even if it does not endanger his life. For this accident an ice-bag to the epigastrium, and the use of astringents, such as Monsel's solution, silver nitrate, tannin, and adrenalin are indicated; and the pill mentioned above of lead, camphor, and opium may be given; rectal feeding should of course be substituted for feeding by the mouth. The methods just noted are better adapted to control hematemesis than the use of sulphuric acid

or of turpentine, formerly much employed for this purpose. Sensory disturbances, such as lightning-like pains in the extremities, like those of tabes, are a leading symptom in many cases of Addisonian anemia. For these disturbances the narcotics never should be used except in the case of an acute emergency. It is much better to apply a snugly fitting flannel bandage, with rest and elevation of the painful limb, and to massage the parts with some anodyne rubefacient such as methyl oleosalicylate.

### LEUKEMIA.

Leukemia as a clinical entity bears many of the hall-marks of an infectious process, affecting selectively the bone-marrow, the spleen, and the lymphatic apparatus of the body. In the more active varieties of the affection are those similarities to an infection the more conspicuous, as evidenced by the prominence of hyperpyrexia, profound asthenia, spontaneous hemorrhages, and splenomegaly as prominent clinical features of such examples of the leukemic process. As to the exact nature of the infectious principle at work, there is no available information. It may be some unidentified specific micro-organism, or it may be a multiple infection; at all events, the process, whatever its real character, is generally characterized by a clinical course of well-marked chronicity, likely to become interrupted from time to time by acute exacerbations which sooner or later prove fatal. Other cases lack this chronic character, and exhibit from the first an active violence of the clinical picture which resists all the efforts for improvement at least temporarily effective in the leukemias of less fulminant type.

Here may be mentioned the inherent tendency of leukemic blood suddenly to undergo an aleukemic transformation under the influence of some one of the intercurrent infections, such as pneumonia and diphtheria. The apparently close interrelationship between various infections, and the appearance of relatively large numbers of myelocytes in the circulating blood, gives further food for thought on the question of leukemia's infectious nature; and the same comment applies to the seemingly selective action of tonsillar and intestinal infections upon the myelogenous tissues.

With this idea in mind, it is still possible, for the sake of convenience, to designate by the predominant features of the blood picture two different types of leukemia, the *myelogenous* and the *lymphatic*, and further to speak of two wholly artificial clinical varieties of the disease, the *acute* and the *chronic*. It is sufficient here to state that of these forms of the disease the myelogenous is more likely to follow a more chronic course than the lymphatic, and that, as a rule, it is more radically affected by therapeutic measures, which unfor-



Fig. 1.—Leukemic enlargement of the spleen. (From Da Costa's Physical Diagnosis. Copyright, W. B. Saunders Co.)

tunately promise little more than a temporary amelioration of the symptoms in either form of the disease.

In the management of a case of leukemia, irrespective of its exact clinical variety, there are several distinct lines of treatment to be followed, dealing with the blood picture, with the general symptom-complex, and with special individual symptoms of the leukemic state.

The *blood picture* in all forms of the disease is alike, in that it is characterized by an excessive number of leucocytes in the circulating blood, and by a hemoglobin deficiency and loss of

erythrocytes, which frequently accounts for an anemia of the most extreme grade.

The differences in the blood pictures of the two clinical varieties of leukemia may be briefly summarized by associating with the myelogenous type the blood changes known as *myelemia* and with the lymphatic type those referred to by the term *lymphemia*.

In *myelemia* the leucocyte increase is made up predominantly of myelocytes, which constitute approximately one-fifth of the total number of leucocytes, ordinarily forty or fifty times higher than the accepted normal count.

The myelocytes, which are regarded as the immediate precursors of the normal polynuclear neutrophiles, are the product of an imperfect and overstimulated hematopoiesis, by fault of which the bone-marrow produces large numbers of these immature cells of variable size, with a single eccentric non-convoluted nucleus deficient in chromatin network, and a protoplasm crowded with delicate neutrophile granules. In health, such cells, as they age, gradually diminish in size, acquire a denser nuclear structure, and show an increase in the number of protoplasmic granules whose neutrophilic reaction persists unchanged, until finally they become transformed into normal polynuclear neutrophiles.

The metachromatic mast cells also are very numerous in the myelogenous type, the eosinophilic leucocytes (both polymorphonuclear and myelocytic) occur in abundance, while widespread degenerative changes affecting the protoplasm and the nuclei of all the white corpuscles are conspicuous features of the stained specimen.

In *lymphemia* the high leucocyte count is due to a disproportionately excessive number of lymphocytes, which, as a rule, constitute more than nine-tenths of the different varieties of leucocytes. In the more chronic lymphatic leukemias, the small lymphocyte generally is the prevailing type of this cell encountered, whereas in the acute forms of the disease the larger varieties of lymphocyte predominate. Myelocytes are present in fractional percentages in lymphemic blood, and the same is true of mast cells and of eosinophilic leucocytes.

Aside from the essential leucocyte proliferation of leukemia, the associated anemia is an important detail of the dis-



ease, and in some instances this feature is so conspicuous as to deserve quite as careful management as the underlying leukemic process. Other conditions being equal, the more acute the type of leukemia the more severe the attendant hemoglobin and erythrocyte losses, and the greater their tendency to conform to a progressive, pernicious type.

In contrast to the more moderate grade of anemia commonly found in chronic myelogenous leukemia, the blood deterioration in acute lymphatic leukemia is likely to reach an extreme grade, and to show, in consequence of the modified embryonic blood manufacture, numerous erythrocytes of the fetal type, designated as megaloblasts and mesoblasts, together with many nucleated erythrocytes, termed normoblasts, also met with in other severe anemic conditions. The leucocyte count tends to remain at a lower level than is the rule in myelogenous cases.

In both forms of leukemia the effect upon the leukemic blood picture of complications such as hemorrhage, diarrhea, and intercurrent infections should be recalled; the first-named conditions provoking an exaggerated hemoglobin and erythrocyte loss with polymorphonuclear neutrophile leucocytosis; the second, a moderate and transient improvement of the anemia due to blood concentration, and the last a temporary subsidence of the original leukemic blood findings.

### TREATMENT.

So long as the attendant anemia does not progress acutely, the leucocyte count is not subject to violent exacerbations, and the splenomegaly remains within reasonable limits, it is questionable whether any form of active treatment should be instituted in a patient affected with chronic leukemia, either of the myelogenous or the lymphatic variety. This temporizing attitude is prompted by the tendency of this disease suddenly and without warning to suffer violent relapses just at a time when a permanent cure is looked for—relapses which carry the blood deterioration far below the level originally determined; and which have proved rapidly fatal in not a few instances.

With this unfortunate peculiarity in view, the physi-

cian proceeds with those measures aimed to overcome the three chief existing morbid processes of the average leukemic subject: the active output of pathologic leukocytes, the huge splenic, hepatic, and lymphatic tumors, and the severe progressive loss of hemoglobin and erythrocytes. For the correction of these symptoms, present practice allows the choice, singly or in combination, of radiation with the Röntgen ray, the administration of benzol, and the judicious use of hematinics, such as iron and arsenic, supplemented by a generous dietary of nitrogenous meats, and various ferruginous foods. Further details of the case management are discussed after the consideration of these therapeutic ventures.

In chronic myelogenous, and, with less certainty, in lymphatic leukemia, the therapeutic use of the *Röntgen ray* has become an established means of controlling the hyperactivities of the leukopoietic tissues, and of destroying the excess of circulating leucocytes. But against its advantages the clinician must weigh certain disadvantages inherent to the  $x$ -ray, since to overradiation may be attributed distressing skin burns and also a fatal type of leucocytotoxemia, while under-radiation occasionally provokes a stimulation of leucocyte production by fault of which the leukemic process flares up in an astonishing manner. To protect the patient from the first of these unfortunate by-effects, the repeated exposure of the same part of the body should be avoided, by mapping out the surface over the spleen, the long bones, and the superficial lymphatic glands into a number of exposure areas upon which the  $x$ -rays are focussed in rotation according to a prearranged plan. It is also customary to make use of a covering of lead plaster or of sheet silver as a protective shield to filter out the irritant and therapeutically inert rays without interfering with the action of the curative rays. Barring the ever-present personal equation, and controlled by frequently repeated blood examinations, radiations of five, ten, or twenty minutes' duration, varying from one *séance* each day to one weekly, will be found satisfactory in most instances.

Whatever be the routine adopted, radiation should not be pushed too rapidly for fear of exciting a reaction which may fatally intensify the leukemic process, and safety demands initial exposures of brief duration at infrequent intervals—

details obviously to be determined by the peculiarities of the individual case.

Fever, symptoms of acute intoxication, the development of additional leukemia tumors, prostration, cachexia, and anemia characterized by extreme oligocythemia and numerous megablasts forbid the use of the Röntgen ray. Renal complications do not absolutely contraindicate this form of therapy, but are a danger signal to pursue it with exceeding caution.

To sum up the utility of radiotherapy, it may be regarded as a means of arresting a large proportion of early cases of myelogenous leukemia, and of improving the blood picture and clinical symptoms of many advanced ones. In acute leukemia little benefit can be anticipated from this or from any other form of treatment, and in such instances arsenic and thorium-*x* (*q.v.*) offer more hope of prolonged life. Lymphatic leukemia is less susceptible to *x*-ray therapy than the myelogenous variety, and recurrences, so common in both, are more frequent and more fulminant in the former. Warthin's studies<sup>25</sup> demonstrate the pathologic basis of the foregoing statement, and prove that the action of the *x*-rays, although it modifies the leukemic process by exciting inhibitive degeneration, leaves the essential leukemic lesions to progress virtually unchecked. Indeed the leucopoietic tissues may suffer complete destruction, with a resultant aleukemic condition of the blood, and, after a lapse of time, with the growth of considerable undifferentiated leukoblastic tissues, and perhaps a consequent return of the leukemic blood changes. Symptomatic cures have been reported, some of several years' standing, but on the whole one must not regard radiotherapy as specific, although it fills an indispensable place in a selected class of cases.

Under such a regimen, carried on for ten days or a fortnight, the high leucocyte count tends to fall normalward, and the pathologic varieties of cells to diminish; simultaneously or shortly afterward the enlarged spleen and liver become smaller, and the palpable lymphatic glands soften. Very significant histologic changes in the cells of the circulating blood include nuclear swelling and chromatin fragmentation affecting the lymphocytes, the polymorphonuclear neutrophiles, and their pathologic marrow antecedents, the myelocytes.

Finally, the protoplasm of the granular cells become studded with vacuoles and devoid of granules, and in successful cases the myelocytes and mast cells disappear from the peripheral circulation. Definite improvement in the hemoglobin percentage and the erythrocyte count later appears, to complete the histologic evidence of the blood regeneration thus induced by röntgenization.

*Benzol* must be used with caution, for its action is that of a leucocytotoxic agent, and in an overdose in a susceptible subject it may provoke intense renal inflammation, free hemorrhages, extreme anemia, and striking leucopenia. The drug's activities are essentially those of the impure benzene of commerce, and are directed primarily upon the leukoblastic tissues, whose functional activity in both normal and abnormal states is markedly diminished. Von Korányi,<sup>26</sup> who suggested this form of hemotherapy, based his idea of its clinical application on Telling's report, in 1910, of his experiments to demonstrate the leucocytotoxic action of the drug. The pioneer work thus begun was soon supplemented by other Continental clinicians, notably by Kiralfi<sup>27</sup> and in America by Billings.<sup>28</sup>

In the favorable case benzol causes a moderate preliminary rise in the number of leucocytes, followed by a striking diminution of their number, particularly affecting the polymorphonuclear neutrophiles. The pathologic types of leucocytes more or less rapidly diminish, and as the myelocytes, mast cells, and other distinctively leukemic cells disappear, a virtually normal different leucocyte count is approached. Coincidentally there is usually a moderate, sometimes decided, hemoglobin and erythrocyte fall, although but rarely does the associated anemia demand suspension of the treatment. As a rule, these blood changes are attended by a decided improvement of the patient's health and strength, and by rapid diminution of the size of the spleen and liver, with less conspicuous disappearance of the enlarged lymphatics.

The ill-advised use of the drug results in a total atrophy and destruction of the hemogenetic organs, total loss of coagulation, and an aleukemic blood picture associated with a high-grade anemia.

From this it would appear that benzol, despite its real



value in the symptomatic cure of the leukemias, must be regarded as a powerful leucocytic poison, possessed of a toxic action which when once excited cannot be neutralized. Another drawback to its indiscriminate use is its variable activities in different individuals, a defect to be explained chiefly on the ground of great differences in personal susceptibility to its action.

Gastric disturbances, headache, vertigo, bladder irritability, progressive anemia, and urine changes indicative of renal irritation should be regarded as signs for the immediate withdrawal of the drug, the action of which, it should be noted, tends to persist for some time after its use has been discontinued. These untoward effects of benzol, according to the consensus of opinion, are more likely to appear in walking patients than in those confined to bed during its administration; hence its use is safer in the hospital ward than with out-patients.

The most successful method of administering benzol is in freshly filled gelatin capsules, with an equal quantity of pure olive oil, the initial daily dose of the drug being 30 grains (2 Gms.), with 45 grains (3 Gms.) the second day, 60 grains (4 Gms.) the third day, and 75 grains (5 Gms.) the fourth day, this maximum dose of 5 grams being continued each day, provided that no ill-effects arise, until the leucocyte count has fallen approximately to as low as 20,000 cells per cubic millimeter.

On the whole, benzol, if used intelligently in carefully selected cases, offers more rapid and more certain effects than any other means at the internist's disposal in the management of the chronic leukemias. It should quite replace the huge doses of Fowler's solution of arsenic formerly given; it is certainly more effective than salvarsan; and not only more efficacious but cheaper and less elaborate than the employment of the Röntgen ray, useful as may be the last-named when skilfully applied.

This fact must be emphasized: that benzol, contrary to first impressions, cannot be looked upon as a radical cure for leukemia. The most that can be hoped for, despite the dramatic disappearance of the leukemic blood picture and the subject's obviously bettered condition, is a temporary remis-

sion of the symptom-complex, which, unless a prolonged benzol therapy is persisted in, results at best in a lease of life measured more commonly by months than by years. Pushed too far, benzol produces a fatal breakdown of the blood-forming organs, and, on the other hand, too moderate a dose results in violent stimulation of the leucoblastic tissues, and only exaggerates the leukemic process. Patients do better on a treatment of benzol combined with  $x$ -ray therapy than on benzol alone.

*Thorium-x*, which possesses radioactive properties, has been used to some extent to control the abnormal leucocytic output of leukemia, owing to its pronounced selective action on the leucocytes in this disease. This action is similar to that exerted by intense radiation with the  $x$ -ray, and to that produced by radium emanations. The metal is usually administered internally, at intervals of two or three days, in doses of from 75 to 150 electrostatic units, until its effects (*v.i.*) are apparent; or a single intravenous injection of 5000 units may be given, although this is more risky and may set up an extreme erythrocytic destruction.<sup>65</sup> In the limited number of instances in which the thorium salt has been used, satisfactory leucopenia, improvement of the different leucocyte count, diminution of the splenohepatic tumors, and softening of the lymphatic enlargements have promptly followed, although at the present writing the permanence of these changes cannot be unequivocally attested.

In favor of the thorium treatment are its harmlessness under intelligent administration, its exact and graduated dosage, and, as contrasted to treatment by intense radiation, the avoidance of injury to the skin from  $x$ -ray burns. Most of the reported thorium "cures" have been in cases treated apparently without success with the Röntgen ray, hence confusion must exist as to the real benefit derived. This, however, should be conceded: thorium seems to supplement the good effects of radiation, and the two methods of treatment may be tried in combination to effect the sought-for permanent leucocyte decline.

*Arsenic*, formerly quite generally in vogue for its reputed curative properties in leukemia, has been abandoned in its former rôle, but still is of distinct utility in combating the

concomitant anemia, and as a substitute for, or an accompaniment of, radiation and the newer drugs, benzol and thorium.

Arsenic is indicated in patients who, after a comprehensive course of one of these therapeutic measures, either fail to improve, grow distinctly worse, or exhibit a distressing intolerance. In such instances it is sometimes possible to discontinue the plan of treatment first pursued, and to substitute in its place a vigorous course of arsenic, which later may or may not be combined with the original treatment, as circumstances determine the wisdom of such action. It is also customary to resort to arsenic as a post-leukemia regimen in cases symptomatically free from the disease, and to use this drug freely in examples of the aleukemic condition induced by intensive treatment by other methods. Fowler's solution (*Liquor potassii arsenitis*), if no personal hypersusceptibility exists, can be relied upon for prolonged administration and in leukemic cases it acts quite as well as the more expensive salvarsan and sodium cacodylate. The initial doses, 3 to 5 minims (0.18 to 0.30 mil) daily, should be pushed by increasing 1 minim (0.06 mil) over the preceding dose on alternate days until a daily intake of approximately 15 or 20 minims (0.92 or 1.25 mils) is attained, at which dose it should be continued for an indefinite period. The old method calling for amazingly large doses of arsenic is now known to be a harmful routine for the leukemic subject, and has been abandoned for conservative blood building without risk of arsenic poisoning. Should Fowler's solution in adequate dosage prove intolerable, it is sound therapy to employ arylarsonates, of which atoxyl is one of the most useful preparations. Administered hypodermically, in daily doses of from  $\frac{1}{2}$  to 1 grain (0.032 to 0.065 Gm.), the drug should be given for three consecutive weeks, and the injections suspended the fourth week; by this method the hematinic action of the salt is actively enlisted, without fear of toxic effects either direct or cumulative.

Those who have used atoxyl intelligently are struck with its inefficacy after an initial improvement of the blood picture, which, as a rule, consists of a moderate, although distinct, decline in the number of leucocytes and percentage of myelocytes and mast cells. When this point is reached (and

unfortunately it is a point far short of the normal level), continuance of atoxyl does no good whatever, nor do toxic evidences of arsenic supervene.

If sodium cacodylate be chosen, this arsenic salt should be used by intramuscular injection in doses of 2 grains (0.13 Gm.) three times weekly.

To counteract the extreme hemoglobin deficiency existing in leukemic subjects, the persistent and free use of iron is called for, to supplement the more dramatic results achieved by one of the leucocytotoxic agencies. Recalling that this grave blood disorder is inherently a form of myeloid hyperplasia closely related to, if not actually part and parcel of, a malignant infection, the essential importance of this metal in an endeavor to combat the associated anemia is apparent. Three principal factors are accountable for this high-grade leukemic anemia: faulty hematopoiesis, attributable to the leukemic bone-marrow lesions; hemolysis excited by the inroads of the circulating toxins; and interference with food absorption and assimilation. Factors such as these adequately explain the presence of leukemic anemias, always of high grade, and frequently tending to become characterized by the histologic hall-marks of a grave type of essential blood deterioration.

As a rule the hemoglobin deficiency in leukemia is much more decided in the lymphatic type than in the myelogenous, and, irrespective of the type of the diseases, tends to be more decided in acute than in chronic cases. The most conspicuous feature of a leukemic anemia is the prevalence of normoblasts and in the acute forms of the disease the tendency of the erythrocytes to undergo structural changes of a most pronounced nature, which in some instances at least reminds one more than superficially of the blood picture incident to true Addisonian anemia.

So long as the iron salt chosen is well tolerated and efficient, it matters but little what form of iron is given to abate the leukemic anemia. Inasmuch as in giving iron to the average leukemic patient means a long siege of treatment, the endeavor must be made to use one of the less irritant forms of the metal, and one in which confidence may be placed for the consistent improvement of a deficient hemoglobin percentage. The iron carbonate recommended for the treatment of



chlorosis, citrate of iron, and ferratin all may be taken for some length of time without unfavorable effects, and all are satisfactory hematinics.

The intramuscular injection of *defibrinated blood* has been suggested as rational therapy in leukemia, but the procedure has not been carried out with favorable results, save in isolated instances. Kiralfi<sup>29</sup> advises such treatment especially in leukemias in which, after a course of radiation and benzol, the initial improvement has been succeeded by extreme leucopenia and grave anemia. In such cases the injection of 10 mils (2.7 f3) of fresh human blood into the gluteal muscle apparently causes a fall of the leucocyte count to the mean normal maximum and by a definite improvement in the hemoglobin and erythrocyte figures. *Pari passu* with the betterment of the blood picture the other clinical features tend slowly to improve, but not, so far as can be determined, to the point of an actual cure of the leukemic condition. The intramuscular injection of blood, it would seem, is called for, not as a factor of eradicating the underlying leukemic process, but rather as a means of controlling the acute leucocytolytic flarebacks which so frequently attend the active use of the Röntgen ray or benzol therapy.

Aside from the foregoing more or less accepted methods of treatment, the therapeusis of leukemia, in common with other incurable maladies, entails the trial of a long list of reputed remedial measures, none of which can be regarded as permanently helpful.

Thus the injection of *bacterial toxins* has been largely advocated, on the premise that leukemic processes are frequently interrupted and temporarily held in abeyance by various intercurrent infections. Larrabee,<sup>30</sup> arguing that the deliberate injection of bacterial toxins should have a similar effect, treated several cases with Coley's fluid (*Streptococcus prodigiosus* toxins), with equivocal results; and Baldauf,<sup>31</sup> reports fair success with the use of bacterial toxins in myelogenous, but not in lymphatic, types of the disease.

*Tuberculin injections* in the treatment of leukemia have been attended by little or no permanent improvement, possibly because, as Dock<sup>32</sup> suggests, tuberculosis complicating a leukemic process has no effect in modifying the latter, such as an

intercurrent streptococcemia does. Alien leukemic serum has been tried by Capps,<sup>33</sup> splenic extract by Jacobs,<sup>34</sup> formalin by Baily,<sup>35</sup> and cinnamic acid by Richter,<sup>36</sup> all with disappointing results in so far as the actual control of the leukemic process is concerned.

*Splenectomy*, despite its utility in splenic anemia, and in some examples of Addisonian anemia, is emphatically contraindicated in leukemic subjects. The futility of this operative procedure in all types of this grave blood disorder is well expressed by von Leube,<sup>37</sup> who insists that "the time has come to discontinue all efforts to cure the disease by injections . . . into the spleen, by faradization or galvanopuncture of the organ, by extirpation of the glands or by splenectomy."

Of the numerous *special symptoms* which are prone to complicate a leukemic condition, gastro-intestinal disturbances, cardiac disorders, and various forms of dropsy are conspicuous examples, and often are present in such a striking and stubborn form as to defy all attempts for their amelioration.

Of the *gastro-intestinal disturbances*, obstinate diarrhea is a prominent symptom, and one frequently most difficult to control. It arises from leucocytic infiltration of the intestine, swelling of the lymphoid follicles, and in some cases ulceration of the wall of the gut, which lesions cannot be expected to yield to the measures used for the control of an ordinary simple intestinal catarrh. These complications, depending, as they do, upon the underlying leucocytic process, may be treated with a view to the emergencies they provoke. Opium in full doses, for example, and frequent irrigation of the bowel with enemas of iced water, of alum (2 per cent.) of tannic acid (2 per cent.), of Monsel's solution (5 per cent.), of argyrol (0.5 per cent.), are useful for the control of the diarrhea; or albuminate of tannin in 15-grain (1 Gm.) doses, and a pill containing 1 grain (0.65 Gm.) lead acetate, 2 grains (0.130 Gm.) camphor, and  $\frac{1}{4}$  grain (0.016 Gm.) opium, is useful as a supplementary measure for the same purpose. Rest in bed, restriction of the diet, and the use of lactobacillary products with pancreatin, diastase, and pepsin are helpful measures, though not *per se* curative. Loss of appetite, nausea, vomiting, and other purely gastric symptoms, are to be treated along general lines.

*Cardiac disturbances* more commonly are attributable to the pressure of the huge splenic tumor than to organic lesions of the heart's musculature or endocardium. If due to the splenomegaly, the use of a bed-rest, to afford a semi-recumbent posture, and the habitual avoidance of the dorsal decubitus by the patient, will go far to relieve the discomfort. If perisplenitis co-exists, as not infrequently is the case, these mechanical measures may have to be supplemented by the use of some form of opium, and by the application of adhesive strips adjusted so as to support the splenic enlargement and to limit the respiratory excursions of the lower left thorax.

In the presence of an actual cardiac lesion, which if it exists, commonly consists of fatty changes in the musculature, strychnin, digitalis, and strophanthus are capable of much good, if used according to definite indications. Syncope and paroxysms of dyspnea, most common complicating symptoms, usually are promptly relieved by some one of the diffuse stimulants such as, for example, Hoffmann's anodyne, spirits of camphor, or aromatic spirits of ammonia.

*Dropsy* is a familiar objective symptom in leukemia, and arises from various factors, which must be clearly identified in order to proceed intelligently with the treatment.

Anemic dropsy is by all odds the most frequent type encountered, and of this sort of edema bogginess of the patient's legs and ankles is the chief symptom. This can be greatly relieved by the firm application of a flannel bandage, and, naturally, by pursuance of the measures originally planned to combat the leukemic anemia.

The edema consequent to the venous obstruction caused by the pressure of the splenic mass is relieved by the use of a flannel binder fitted to the belly, from pubis to xiphoid, so as to afford firm support with upward oblique traction. In splenomegalic dropsy one must also insist on the postural rules recounted above (*v.s.*). Hydragogue cathartics, owing to their irritant effect upon the already diarrheal bowel, should not be employed to relieve the abdomen of a fluid mass, nor is it safe to aspirate the ascites.

Cardiac dropsy, an uncommon complication, calls for treatment directed toward strengthening the tonicity and contractile force of the myocardium, therapeutic measures that have been considered elsewhere. (See Myocarditis.)

*Spontaneous hemorrhages* are common, especially in the lymphatic variety of the disease, but, as a rule, the bleeding is not a serious incident, and can be controlled by simple methods, such as firm pressure and the local application of styptics. Commonly the accident amounts merely to epistaxis, or bleeding from the gum; rarely, the hemorrhage is renal, pulmonary, or cerebral; and exceptionally, it is in the form of a cachectic purpura or larger extravasations, particularly in acute forms of leukemia. The treatment of the various factors of the hemorrhages just mentioned is given under the appropriate headings. (See page 52, *et seq.*)

Finally, apart from all routine courses of therapy, originally designed to overcome the essential leukemic disorder, the patient must be enjoined to rest, to live in the open air and sunshine, and to partake of a full and nutritious diet, in an effort thus to postpone the period of bedridden helplessness which ultimately must come in every case. When this occurs the palliative measures, at first mercifully helpful, prove of no avail whatever, and one must resort to the judicious use of opiates, to deaden the effects of the leukemic inroads and to tranquilize the sufferer's last days. In acute leukemia the average tenure of life, from the onset of the initial symptoms, may be not longer than a few weeks; in chronic cases, it is approximately three years, and sometimes longer.

### CHLOROMA.

Chloroma is to be regarded as a morbid transitional lesion between true leukemia and malignant neoplasms, as signified by its synonymous terms, *green cancer* and *chlorosarcoma*.

Pathologically, this disease is characterized by neoplastic growths of greenish color implicating especially the orbits, temporal fossæ, and vertebræ; and by marrow changes similar to those of myelogenous leukemia. The blood picture is inconstant, but, as a rule, it typifies lymphemia, with large hyaline mononuclear cells in excess; or the myeloid type of blood may prevail.

The *treatment* of chloroma can be but palliative, for the disease invariably runs a rapidly fatal course, lasting not longer than six months, in the average case.



The *x*-ray may afford a temporary amelioration of the symptoms, and the vigorous use of iron and arsenic occasionally is of similar service, but no therapeutic measures avail in permanently controlling the widespread chloromatous infiltration, once its invasion of the subject's adenoid, visceral, and osseous structures is excited.

### HODGKIN'S DISEASE.

Hodgkin's disease, also known as pseudoleukemia and lymphadenoma, is a condition of obscure origin and unknown etiology, featured clinically by progressive enlargement of the



Fig. 2.—Generalized glandular enlargement in Hodgkin's disease. (From Da Costa's Physical Diagnosis. Copyright, W. B. Saunders Co.)

lymphatic glands and usually of the spleen in association with an anemia of variable degree. As a rule, the actual blood picture is but little altered, but in some cases the deterioration is excessive. The glands commonly affected are those of the neck. Appearing in this site, or perhaps in the glands of the axilla or of the groin, the diagnosis may be more readily made than when the condition arises in the retroperitoneal glands or in the peribronchial lymphatic structures; under the latter circumstances the diagnosis ordinarily is made at autopsy. Hodgkin's disease is to be differentiated from other conditions which produce enlargement of the lymphatic glands, first

among which is tuberculous adenitis, while other conditions demanding careful differentiation are lymphosarcoma, lymphocarcinoma, and chronic leukemia. In Hodgkin's disease the enlargements are usually discrete, and the glands rarely tend to soften, coalesce, suppurate, and fistulate, as is the common course of a tuberculous adenitis. Hodgkin's disease is progressive in course, while tuberculous adenitis may remain quite stationary and is continued over a longer period of time. Lymphosarcoma, on the other hand, extends much more rapidly than either of these lesions, and soon implicates surrounding tissues *en masse*. Carcinoma of the lymphatic glands is associated with the cachexia and pain so often characteristic of cancer elsewhere in the body. Chronic leukemia, of course, presents a profoundly altered blood picture. Hodgkin's disease may be characterized by an irregular fever. Should the glandular enlargements be of such size as to press on surrounding structures, pressure symptoms ensue; decided swelling of the arm may result from this cause, as also may dyspnea, gastro-intestinal symptoms, and syndromes referable to the nervous system. Bronzing of the skin may appear when the abdominal glands are invaded, and a purpuric rash may be present at times.

The blood, while appearing thin and pale, does not show much departure from the normal picture. The erythrocytes may be slightly diminished in number, and the leucocytes rarely are above the normal maximum limit of 10,000; the hemoglobin is, however, reduced, 15 or 20 per cent. The disease would seem to be more common in males, judging from Gower's studies of 100 cases, which showed that 75 men were affected to 25 women. The prognosis is extremely grave. The average duration of life after the condition is diagnosed has been placed at two years—a statement which, of course, is modified by the previous condition, life, and history of the patient, and by the response of the individual to treatment.

#### TREATMENT.

The first step in treatment is to arrive conclusively at the correct diagnosis of Hodgkin's disease by the exclusion of a possible tuberculous glandular enlargement. If information in this regard cannot be obtained from tuberculin reactions, such

as the von Pirquet test, it is quite permissible to excise a small portion of a gland for histologic study. Hodgkin's disease often yields temporarily to the use of *arsenic*, such as Fowler's solution, in gradually increasing dose, until perhaps 15 or 20 minims (1 to 1.3 mil) have been reached, or until the physiologic tolerance of the drug is exhibited by a metallic taste in the mouth, by puffiness under the lower eyelids, or by the onset of gastro-intestinal symptoms of moderate degree. Of late, arsenic in the form of salvarsan injections has been happily employed. Following such intravenous injection, the glands have been observed to shrink and harden, the temperature to drop, and the patient's general condition to improve. Sooner or later, however, the symptoms will recur, and subsequent injections are not accompanied by the gratifying results which follow the first administration of salvarsan.

*X-ray therapy*, administered at the hands of a competent operator, is always to be thought of and tried. When painful or urgent symptoms arise as a result of glandular pressure, operative interference is, of course, indicated to relieve the discomfort.

To a limited extent *vaccine therapy* enjoys a certain vogue in the treatment of Hodgkin's disease, but, unfortunately, without either curative or even palliative effect, to judge from the moderate number of cases available for study. Even autogenous diphtheroid vaccines, such as used by Smoot and Carrell,<sup>38</sup> apparently prove futile, for their use is followed merely by a temporary decrease in the size of the enlarged glands, with no permanent improvement of the local lesions or of the constitutional symptoms.

The treatment of Hodgkin's disease by the injection of *tuberculin* seems wholly empiric and unjustified. This means of therapy is based upon nothing more tangible than the fact that the coincidence of tuberculosis and pseudoleukemia in the same individual may modify the latter process.

The *surgical treatment* of Hodgkin's disease is warmly advocated by Yates and Bunting,<sup>66</sup> who, assuming the part played by infection, urge as a preliminary step the thorough elimination of all portals of entry for invading bacteria, by attention to inflamed tonsils and infected teeth and accessory sinuses. This accomplished, radical extirpation of all diseased

lymphoid tissue within reach of the knife is done, and this is followed by systematic radiation and by the use of immune serum. These authors consider this routine exceedingly useful, believing that it is curative in fully 20 per cent. of all cases.

Admitting the possible relationship of Hodgkin's disease and sarcoma, it is but natural to urge the adoption of identical surgical measures in both conditions, at least in the former's incipency, at a time when a single gland is affected or when the adenopathies have not progressed far. The operation should be radical, and, as Coley<sup>39</sup> points out, must include enucleation of the tonsils, if these glands, the probable seat of the primary infection, also are enlarged. Of Coley's series of 22 cases,<sup>40</sup> treated variously by surgery, *x*-rays, and bacterial toxins, 13 died, 2 showed no improvement, 4 definitely grew better, and 3 were lost sight of.

The use of an *immune serum* prepared with the *Bacillus hodgkini* is advised, on the basis of the specificity of this germ, and in subjects thus treated a fall of temperature, lessening of the glandular swellings, and improvement of the subjective symptoms have been observed by several investigators.

The general condition of the patient should be appropriately treated. Tonics, to support the failing strength may be required, among which iron, quinin, and strychnin may be advantageously used. Codliver oil or other emulsified fats may be prescribed with good effects. The use of antipyretics is rarely justified, the fever being better combated by the use of tepid baths and similar harmless measures. The condition of the intestinal canal, which may be readily upset by torpor of the liver, will require attention. Anorexia and nausea call for the exhibition of simple digestants. For the profound prostration and symptoms of myocardial degeneration digitalis in supportive doses is necessary.

### SPLENIC ANEMIA.

An excellent description of the disorder under discussion is expressed by the words of Sir William Osler: "Provisionally, it is useful to group together cases of idiopathic enlargement of the spleen with anemia without lymphatic involvement, and to label the condition splenic anemia."<sup>41</sup> This grouping fur-



nishes a fairly definite working basis for the investigation of the loose term, splenic anemia, from a clinical viewpoint, but it does not indicate the dominant pathologic peculiarities of the various types of the affection. It excludes the splenic enlargements so common in malaria, tuberculosis, syphilis, secondary anemia, and those incident to leukemia, Hodgkin's disease, pernicious anemia, and tropical splenomegaly. It includes certain anemic splenomegalies of peculiar character, the three principal varieties of which are designated as Banti's disease, splenomegaly of the Gaucher type, and von Jaksch's splenic anemia of infancy.

The chief pathologic changes of adult splenic anemia relate to enlargement of the spleen, and to secondary cirrhosis of the liver. To a less conspicuous extent there are, more or less constantly, definite changes affecting the gastrointestinal tract, the portal circulation, and the bone-marrow.

In the ordinary type of splenic anemia the splenic enlargement is referable to a generalized fibrosis and hyperplasia shared in common by the *réticulum*, capsule, and malpighian bodies, in which process proliferation of the blood sinus endothelial cells is an important detail. By some investigators this endothelial proliferation is considered the characteristic pathologic change, and one accounting for the elaboration of a hemolytic toxin to which the coincident anemia is directly due. Associated with these changes, moderate fibrosis of the portal areas of the liver, thrombosis of the portal vein, and enlargement of the hemolymph glands are conspicuous, but inconsistent, secondary lesions. The bone-marrow undergoes no distinctive alteration; the lymphatic glands are unaffected.

In certain cases of splenic anemia, the complete syndrome of anemia, splenomegaly, hepatic cirrhosis, and ascites develops as a terminal stage, and to this group the term Banti's disease is applied. The liver, after a temporary increase in its size, corresponding to the acute stage of the disease, undergoes cirrhosis greatly resembling that of the alcoholic type of Laennec, and from this factor ascites arises. The alterations in the bone-marrow vary in different cases of Banti's disease, lymphoid degeneration, erythroblastic increase, and the presence of phagocyted erythrocytes and blood pigment being the most frequent changes observed.

In the gastro-intestinal tract, general atrophy of the intestinal mucosa, circumscribed thickening of the intestinal wall, and esophageal and hemorrhoidal varices are the lesions whose incidence is fairly constant.

Splenomegaly of the *Gaucher type* represents a clinical entity, characterized by histologic changes wholly different from those already described in the other varieties of splenic anemia, with which it is grouped, more for convenience sake than upon a sound pathologic basis. In Gaucher's disease the splenic enlargement is directly due to an accumulation within the parenchyma of the organ of masses of large endothelial cells, alveolarly grouped, and surrounded by a delicate fibrous network. The cells, which are loosely arranged, vary from 20 to 40  $\mu$  in diameter, contain an abundant almost homogeneous cytoplasm, and have one or more small densely basic nuclei. Similar groups of cells are found in the liver, the bone-marrow, and the lymphatic glands. The exact nature of this cellular invasion of the hematopoietic organs is still a moot point among different investigators, but the general trend of opinion fixes their origin as endothelial.

The clinical diagnosis of splenic anemia is based upon the leading features of the syndrome such as the idiopathic splenomegaly, a hemorrhagic tendency, and a well-defined anemia attended by disproportionate hemoglobin loss and by distinct leucopenia with relative lymphocytosis. The course of the disease, furthermore, is unduly prolonged, and the subject shows evidences of symptom-groups variously referable to the anemia (asthma, dyspnea, vertigo, cardiac palpitation, hemic murmurs, edema); to the hemorrhagic diathesis (epistaxis, hematemesis, hematuria, petechia); and to the splenic tumor (pain, distension, ascites). To these principal findings must be added the absence of enlarged superficial lymphatic glands, and, in the Gaucher type of the disease, the presence of an appreciable increase in the size of the liver.

#### TREATMENT.

One of the first essentials of treatment is provision for the patient of a proper hygienic environment, with all that the term implies relating to fresh air, rest, and a nutritious palatable dietary.

The knife affords the only radical cure for splenic anemia, and it is an acknowledged fact that in approximately 70 per cent. of early cases splenectomy are followed by a rapid disappearance of the anemia and the other features of the disease and finally by a complete and permanent cure. Particularly favorable is the outlook when the operative procedure is preceded by transfusion,<sup>42</sup> and in cases recognized before the disease has worked its widespread systemic inroads. Unfavorable in prognosis are all intense grades of anemia, excessive enlargement of the spleen and liver, and the super-vention of the changes incident to Banti's disease. W. J. Mayo<sup>43</sup> and Miller<sup>44</sup> have reviewed the surgical treatment, and the reader is referred to this work for authoritative data on the subject.

The x-rays also have been used, more as a palliative than as a curative measure, for although systematic radiation, such as is practised in leukemia, tends to diminish the size of the spleen, and in general to improve the other symptoms, this method of therapy does little more, and in no sense can be considered curative.

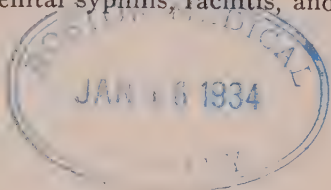
The foregoing remarks also apply to the use of iron, arsenic, and other hematinics, useful as adjuncts to splenectomy, but never an adequate substitute for this operation.

### INFANTILE SPLENIC ANEMIA.

This obscure type of infantile anemia is a doubtful clinical entity, although, for convenience sake, it is permissible to regard it as a form of primary anemia excited by some unknown toxic factor. It corresponds to von Jaksch's "Anemia infantum pseudoleukæmica,"<sup>45</sup> and the "Anemia splenica infettiva dei bambini" of the Italian school.<sup>46</sup>

The disease in question is limited to young children, occurring especially in those of the male sex, and is prone to affect the twins of a family rather than several of the other children. The chief clinical features include acute secondary anemia; high, persistent leucocytosis; and enlargement of the spleen, liver, and lymphatic glands.

With tolerable constancy, prolonged breast-feeding, congenital syphilis, rachitis, and tuberculosis have a more or less



significant causal rôle, although it must be admitted that the exact relation of the foregoing conditions to infantile splenic anemia is conjectural.

The blood changes observed most commonly relate to an unduly low hemoglobin figure with a less striking erythrocyte loss, and a high leucocyte count in which a cellular "heteromorphism" prevails—that is, the excess of leucocytes is largely made up of atypical forms transitional between the two types of lymphocytes, large hyaline cells and myelocytes, and neutrophilic and eosinophilic forms. Large numbers of normoblasts and occasional neutrophilic myelocytes also are a conspicuous feature of the blood picture.

#### TREATMENT.

The treatment of this variety of splenic anemia is essentially the same as that of other blood deteriorations of similar intensity and development, but aside from the free use of appropriate hematinics, preventive measures against various intercurrent infections are urgently demanded, together with the care of the potential factor of the disease, be it lues, rachitis, or tuberculosis. With intelligent management this type of anemia can be arrested in approximately 80 per cent. of cases, according to data given by Rotch.<sup>47</sup>

Iron and arsenic, then, are the directly curative drugs; and codliver oil, olive oil, mercurial inunctions and the rest of the antisiphilitic regimen constitute the equally important correlative measures to be adopted.

For a child two years of age (and splenic anemia rarely progresses untreated beyond this age-limit) it would be appropriate to give, by intramuscular injection, either citrate of iron in 1-grain (0.065 Gm.) doses, daily; or sodium cacodylate in the same amount, on alternate days, in an endeavor thus permanently to increase the hemoglobin and erythrocyte values, and to bring about a subsidence of the high leucocyte counts.

As a supplement to this drug therapy, a generous diet, with a large ration of fats, red meats, milk, and carbohydrates is indicated. (See Rickets.)

The surgical treatment of infantile splenic anemia deserves mention as a possible curative measure, although in no sense



does it play the part that one credits it with in the adult form of this disease. Giffin<sup>48</sup> has studied in detail the results of splenectomy, both in the adult and in the infantile form, and his instructive paper should be consulted by those interested in this method of treatment. The results of the surgical treatment of the infantile form are in no wise comparable to those afforded by the simple use of iron, arsenic, and the other means of blood building noted in a foregoing paragraph.

### PURPURA.

The term *purpura* relates to a symptom, and in no sense refers to a specific disease. The many attempts made in various textbooks to describe purpura as a distinct disease have resulted in a number of sub-divisions and headings which it is not necessary to consider here in detail. The essential morbid process at work in the various types of purpura is a spontaneous hemorrhagic extravasation of blood into the skin, mucous membranes, and viscera.

Ordinarily purpura appears as purple patches upon the dermal and mucosal surfaces, these areas of discoloration usually being discreet, and but rarely coalescent. They are prone to occur, as a toxic evidence, in many septic conditions, and, indeed, it would seem that in the vast majority of instances infection and sepsis are responsible for the symptom. While no specific micro-organism is associated with purpura, the symptom arises not infrequently in bacterial infections such as cerebrospinal ("spotted") fever, pyemia, general septicemia, and pneumonia. The term "cachectic purpura," associated with tuberculosis and Bright's disease, is readily seen to be of an infectious origin; and so is the "neurotic purpura" incident to locomotor ataxia, although here, of course, the *Spirochæta pallidum* is the provocative infection. "Mechanical purpura" occurring in the venous stasis of whooping-cough is now known to be of bacterial origin. Arthritic purpura, which is the "purpura rheumatica" of past medical generations, is now believed to originate from septic foci somewhere in the economy.

The blood picture of purpura suggests the action of some specific poison to the blood plaques, with a coincident hemo-

lysis affecting the number and vitality of the erythrocytes. Thus, in a well defined case, the plaques are unduly scanty; the hemoglobin percentage ranges from 20 to 70; and the erythrocytes vary in number from 1,000,000 to 3,500,000 per cubic millimeter, and show evidences of moderate structural deterioration; the leucocytes, which may be abnormally numerous, are not constantly altered. Hematopexis is, as a rule, delayed, especially in purpurics with striking reduction of the plaque count, although this change is not constant. The so-called "bleeding-time" of the blood in purpura invariably is greatly prolonged. This is estimated by timing the persistence of oozing from a needle prick, which normally ceases within two or three minutes, while in a purpuric it may last for as many hours. (Hayem.<sup>49</sup>) In this connection the reader should consult the recent experimental work of Lee and Robertson<sup>50</sup> on the subject of antiplaque serum and its action on the clotting of the blood.

Purpuric spots vary in size and in color; when small and pin-pointed in size they are called *petechiæ* ("flea-bite"); when large, reaching the size of perhaps 3 millimeters, they are termed *ecchymoses*. First occurring as bright red spots, uneffaced by pressure, they gradually become darker, and eventually fade to brownish stains.

**Purpura Simplex.** This condition arises during the course of frank infectious processes: pyemia, septicemia, and malignant endocarditis produce ecchymoses that may be very abundant. Typhus fever is characterized by a purpuric rash, and the acute infectious diseases of childhood, such as measles and scarlet fever, begin with purpuric spots, and are differentiated by temperature changes, symptoms of constitutional infection, and the subsequent course of the disease. S. Weir Mitchell reported purpuric extravasations following the bite of snakes. In cancer, in Hodgkin's disease, in scurvy, and in the debility of old age purpuric extravasations are frequently features of the clinical picture.

The petechial rash which follows the administration of certain drugs may well be included under *purpura simplex*. Copaiba, quinin, belladonna, mercury, ergot, and the iodids produce eruptions which may be so classified. It is interesting to note that the employes of rubber factories frequently pre-

sent a purpura which is attributed to their coming in contact with benzol, a preparation used as a solvent for rubber.

**Purpura Hemorrhagica.** This condition is known as the *morbus maculosis* of Werlhof. It is seen in frail young girls. After a few days of prodromal languor and lassitude, purpuric spots make their appearance, rapidly increasing in number and size. This eruption—and indeed the same may be said of most purpuric extravasations—is not confined to the surface of the skin alone, but also occurs upon the mucous surfaces of the body; hemoptysis, hematemesis, and bleeding from the bowel (*melena*) may thus induce a serious loss of blood, and should the extravasation take place within the brain, cerebral symptoms and even death may take place.

**Henoch's purpura** is a term employed to describe the severe gastro-intestinal symptoms, pain, vomiting, and diarrhea, which ensue when the rash invades the gastro-intestinal tract, usually in children. *Purpura peliosis rheumatica* is also known as *Schönlein's disease*. This condition, which frequently follows exposure to cold and dampness, is characterized by the association of multiple arthritis, urticarial wheals, and purpuric extravasations. The kidneys are usually implicated, as first evidenced by an albuminous urine charged with tube casts. The majority of persons so affected recover, or they may drag out a miserable and incapacitated existence for a prolonged period and finally succumb.

#### TREATMENT.

The treatment of purpura is a treatment of the underlying condition or disease, of which the purpura is but a symptom. Foci of infections should be sought for and properly relieved. If acute articular rheumatism seems to be the underlying cause, the salicylates are indicated, in dosage varying from 5 to 15 grains (0.32 to 1 Gm.) depending upon the requirements of a given case, or one of the less irritant aspirins may be preferred—*aspirin*, *novaspirin*, or *diaspirin*. If syphilis be the provocative infection, iodids and salvarsan should be exhibited; if pyorrhea, oral sepsis, or dental caries be at fault, such exciting conditions must be relieved. A properly selected diet, excluding those foods which would give rise to gastro-intestinal disturbances, is to be combined with fresh air and a

hygienic manner of living. The anemia which is associated with many cases of purpura indicates the use of iron, preferably in the form of Bland's pill. Fowler's solution (*Liquor potassii arsenitis*) is administered in gradually ascending doses of 1 drop (0.06 mil) a day until the physiologic tolerance of the drug is reached, the dose being then reduced 1 drop (0.06 mil) a day until it reaches the initial minimum administration of 3 drops (0.18 mil). (See Secondary Anemia, p. 4.)

Inasmuch as purpura is often associated with a delay in the coagulability of the blood, calcium lactate may be administered in 5-grain (0.3 Gm.) doses four times daily for a period of three or four days. Calcium chlorid in the same dose is also used in the slow hematopexis of purpurics, with the understanding, however, that both the chlorid and the lactate of calcium, if given too freely, *diminish* the blood's clotting power. Serums or defibrinated blood may be employed by direct infusion, and often exhibit a remarkably beneficial effect where the hemorrhages are of considerable number.

For the profuse bleeding incident to purpuric conditions, injections of gelatin have an essential place in the therapy, inasmuch as this material unquestionably increases the blood coagulability. Preferably the gelatin is used in a solution of 7 grains to 1½ ounces (0.46 Gm. to 45 mls) of sterile water. A single injection of this solution may be sufficient to control the hemorrhage; but there is no contraindication to its repetition as often as necessary. Thus, gelatin is useful in immediately controlling a hemorrhage, while the drug mentioned in the above paragraph is more useful as a prophylactic of this accident.

The injection of human whole blood, in amounts of approximately ½ fluidounce (15 mls) has given gratifying results in many instances. Instead of human blood, horse serum may be used with excellent results, especially in purpura hemorrhagica. Schlenker<sup>51</sup> advises the injection of this agent in ten consecutive daily doses of 2¾ fluidrams (10 mls) each.

In those cases of purpura in which the number of blood plaques is distinctly subnormal, this deficiency may be compensated promptly by the use of one of the newer thrombo-



plastic agents, such as, for example, kephalin or coagulen. The latter, for many reasons, is preferable, and is given, either intravenously, injecting  $5\frac{1}{2}$  fluidrams (20 mls) of a 5 per cent. aqueous solution; or by mouth, giving every fifteen minutes 1 tablespoonful (15 mls) of a solution made by adding .75 grains (5 Gm.) of coagulen to 2 ounces (60 mls) of normal saline solution.

Emetin hydrochlorid, in  $\frac{1}{2}$ -grain (0.32 Gm.) intramuscular injections, is, as a rule, curative in the average example of purpura, but its use must be persistent to obtain this effect, inasmuch as the first few doses but aggravate the symptoms that later disappear under the drug's prolonged exhibition.

On the basis that certain examples of purpura are consequent to disturbances of the suprarenals (loss of vasoconstrictor substance), it is reasonable to prescribe adrenalin in such instances. This animal extract usually is used in the form of adrenalin chlorid in a 1:1000 aqueous solution, of which 5 to 30 minims (0.30 to 1.9 mls) are given by mouth, or by hypodermic injection, several times daily, as the occasion demands. Oil of turpentine is a remedy of good reputation for controlling the bleeding of apparently idiopathic types of purpuric extravasations, being given in 10- or 15- grain (0.65 or 1 Gm.) doses three or four times a day. It is unnecessary to add that any circumstances or any drugs, such as exercise or cardiac stimulants, which cause an increase of arterial pressure, also excite an increase of the extravasations, and therefore are to be avoided.

### HEMOPHILIA.

Hemophilia is a condition restricted to the male sex, characterized by an inherited tendency toward inordinate spontaneous and traumatic hemorrhages. The disorder is attributable to a congenital deficiency in hematopexis, and is also known by the synonymous term, "hemorrhagic diathesis," which seems better descriptive of the condition than is the Greek work "hemophilia," meaning "love of blood." Hemophilia is a very curious and, fortunately, a very rare condition. In order to be classified as a "bleeder," it is quite necessary that one should have exhibited this tendency from

infancy. Injuries of a remarkably trivial nature may, in those of the hemorrhagic diathesis, induce an uncontrollable loss of blood, which in certain instances may prove fatal. A slight blow upon the nose, not of sufficient severity to induce a simple congestion in an ordinary individual, may cause an alarming hemorrhage in the hemophiliac; and the extraction of a tooth is capable of exciting a free hemorrhage whose persistence and extent primarily leads to the discovery of the condition. The loss of blood, however, is not confined to surface injuries alone, but may follow the bruising of a joint, in which event bloody extravasations may produce a hemoarthrosis, while serous hematomas are prone to form subcutaneously and in the muscle sheaths. To these two details of the clinical picture (arthritic effusions and serous hematomas) great diagnostic value is attached. Inconsequential injuries to the mucous membranes may cause intractable hemorrhage.

The physiologic changes which would cause a vessel to continue bleeding after the formation of a clot are not as yet understood; it has, however, been determined that the coagulation time of the blood is much delayed, recent investigations indicating that the normal coagulation time of three minutes is, in the hemophiliac, prolonged to as much as 40 or 60 minutes.

The essential factor of the slow, imperfect clotting of hemophilic blood is still a disputed point among physiologists. It has been attributed by Howell,<sup>52</sup> on most rational grounds, to a reduction of the blood's prothrombin content, presumably consequent to some functional change in the plaques, from which this substance is derived. The recent studies of Hurwitz and Lucas,<sup>53</sup> who found great fluctuations in the prothrombin of hemophilic plasma, attest the correctness of Howell's theory, in contrast to the older premises that a deficiency of thrombokinas, thrombozym, and calcium was the factor of the imperfect hematopexis.

Much has been written concerning the advice proper for the physician to give to those who exhibit this inherited tendency to bleed, and who are contemplating matrimony. Earlier thought on this subject has indicated that the male of a family of bleeders who is himself *not* a bleeder may, with perfect

safety, marry a woman who is not a bleeder; on the other hand, a woman who comes of a family of bleeders, and who is not herself a bleeder, may transmit the family tendency to her offspring, and hence it has not been considered desirable that she should marry. It is not germane here to discuss the moral or religious privilege of the physician to give such purely prophylactic advice on affairs that so intimately concern the happiness of his patient, even though such advice might be heeded; but it is permissible, however, to draw attention to the observations of Sir Lovell Gulland, who informs us that "some family histories have recently been published in which the females and not the males have been affected; and in some of these the transmission has been made through the male side of the house; while in at least one case there has been a reversal, the females having transmitted it in earlier generations, the males in the later."

The induction of hemorrhage in individuals so afflicted may be, as already noted, due to slight injuries, or even may arise spontaneously. In the latter event, a sense of physical well-being and exhilaration frequently precedes the onset of the hemorrhage. Upon the appearance of such aura, it would not be ill-advised to suggest that the patient take a laxative, and that he restrain himself from exercise and emotional excitement, with the view of averting a loss of blood. It is customary with careful surgeons to inquire whether or not this inherent tendency to bleed exists in prospective operative cases; and it would seem to be equally advisable for the dentist to make similar inquiries preceding the extraction of the first tooth.

Certain differences between hemophilia and purpura must be recalled, in order to differentiate the two conditions which, in the typical instance, present contrasting points of discrimination.<sup>54</sup> Hess's studies of the blood in these hemorrhagic disorders show that in the hemophiliac subcutaneous puncture of the skin is rarely followed by an area of hemorrhagic extravasation, while in purpura a consequence of this sort is virtually constant. In hemophilia, moreover, the application of a tourniquet to the upper arm produces no objective sign, but in purpura this procedure causes petechial hemorrhages upon the forearm below the point of constriction. Finally, in

hemophilia great delay of hematopexis and no striking diminution of the plaque count are the rule, in contrast to the slightly prolonged coagulation time and subnormal number of blood plaques in purpura.

### TREATMENT.

The treatment of hemophilia productive of the most brilliant results consists of the use of the various blood serums, of which normal human serum is by far the safest and most satisfactory, chiefly because it contains no foreign protein, and hence the risk of by-effects need not be apprehended. The serum of the horse and the rabbit also are employed, but with these that peculiar reaction known as "serum sickness" must be reckoned with; this alarming consequence occasionally provokes hyperpyrexia and an urticarial scarlatiniform rash with numerous systemic disturbances, occasionally of great intensity.

The serum selected is most satisfactorily administered intravenously, but it may be given subcutaneously, or used in the form of a compress to the point of hemorrhage, if accessible. Under adequate aseptic precautions, from 10 to 20 mils ( $2\frac{3}{4}$  to  $5\frac{1}{2}$  f5) of serum are given intravenously each twenty-four hours until the hemorrhage is under control, or twice this quantity if the subcutaneous method be chosen. In favor of the intravenous route is its immediate effectiveness in supplying normal substances essential to increase a deficient blood coagulability, whereas by the subcutaneous technic the serum reaches the blood stream more slowly, and doubtless is impaired in potency by the action of the tissues from which necessarily it must be absorbed.

Horse-serum may be administered in its original form, or as diphtheritic antitoxin, but in using this foreign serum one must recall the possibility of anaphylactic reactions, and must rule out asthma, hay-fever, and susceptibility to horse emanations on the part of the patient.

In case the transfusion of human whole blood is undertaken as a substitute for one of the serums, the proper preliminary agglutination and hemolysis tests should be made, and, as an additional safeguard, a Wassermann test of the donor's blood.



Purely on empiric grounds, the use of various tissue extracts, internally and locally, sometimes are sufficient promptly to check a hemophilic bleeding. Of these, desiccated thyroid, in doses of from 2 to 4 grains (0.13 to 0.26 Gm.), three or four times daily, is of proved utility, notwithstanding the fact that in non-hemophilic subjects the use of this substance tends, as a rule, to increase the tendency to hemorrhage. Among the other animal extracts recommended for hemostasis, but not so reliable as the one just mentioned, are tissue extracts of the liver, pancreas, spleen, kidney, and ovary. Desiccated suprarenal gland, owing to the potential vasoconstrictor action of its epinephrin content, has been used locally to control hemorrhage, but not with great success.

The various preparations of calcium have a distinct place in the therapeusis of hemophilia, and for a long period have been used because of the more or less well merited properties of this substance to promote clotting of blood having feeble power of hematopexis. Calcium lactate, in doses of 5 grains (0.3 Gm.), three or four times daily, probably gives better results, and is less disturbing to the stomach than calcium chlorid, in similar amounts; or ordinary lime-water, administered in tablespoonful (15 mls) doses with milk, every four to six hours, may be efficacious. Inasmuch as an excess of calcium salts in the blood tends to cause a delay of the coagulation of this fluid, caution in their administration is to be observed, by making frequent hematopexis tests, in order thus to regulate the dosage and length of their administration.

The attendant anemia is to be investigated, and, if well defined or progressive, should be treated on general principles—a bountiful dietary, with a generous ration of milk, fats, and meats, and the prescription of some well tolerated and active preparation of iron, perhaps supplemented by arsenic. (See Secondary Anemia, p. 4.)

In order to control the disorder by affecting the underlying leucocyte deficiency and the defective hematopexis, the administration of nuclein and thymus extract has been suggested by Sir Almoth Wright,<sup>55</sup> who holds that these agencies in combination usually are sufficient to control both the serous and actual hemorrhages incident to this condition.

In the face of an active hemorrhage and the failure of other

hemostatic methods,  $\text{CO}_2$  has been administered by inhalation, on the ground that intravascular coagulation is thereby accelerated. Preferably the purified carbonic acid gas, contained in steel cylinders, is chosen, but in an emergency it may be evolved by improvising a generator made of a large glass bottle with two openings, the lower one covered with a bit of gauze or muslin, and the upper provided with a tight cork, pierced by a stout glass tube, to which rubber tubing is fitted. By filling such a home-made apparatus with chalk, and immersing it into a pan of vinegar,  $\text{CO}_2$  is rapidly generated, and may be fed to the patient by means of the tube.

Should an operation become necessary upon a hemophiliac the coagulation time of the blood may be increased by the preliminary administration of chlorid of calcium in 15- to 30-grain (1 to 2 Gm.) doses three times a day, supplemented if needs be by its use per rectum.

### ERYTHREMIA.

A syndrome distinguished by persistent absolute polycythemia, plethora, cyanosis, and splenomegaly was first described by Vaquez,<sup>56</sup> and subsequently awarded a clinical status by Osler,<sup>57</sup> by whose names the disorder is commonly referred to eponymically. It is also known as polycythemia, qualified by adjectives such as true, myelopathic, cyanotic, and splenomegalic.

In this curious disorder, unlike the polycythemias of physiologic and functional types, the abnormally high erythrocyte count and commensurate hemoglobin figure are habitual and constant features of the clinical picture, which, in addition, is distinguished by the subject's deep cyanosis, difficult breathing, enlarged spleen, tendency to spontaneous hemorrhages, and cerebral symptoms, such as headache, vertigo, and, rarely, apoplectic seizures.

This symptom-group, due to excessive erythroblastic activity of the bone-marrow, arises without apparent exciting cause, and, as a rule, is persistent throughout the clinical course, perhaps marked by periods of transient spontaneous remission. Ordinarily the patient's condition remains stationary for a long period, while others succumb to cardiac failure,

or die with signs of rapid deepening of the cyanosis, or of a complication set up by intracerebral hypertension.

The *treatment* of erythremia, in so far as a cure of the disorder is concerned, is hopeless, for no therapeutic method is available for the control of the essential hemopoietic hyperactivity.

Venesection, to be resorted to periodically, is a means of affording temporary relief of the cyanotic exacerbations, and repeated saline purges should supplement this operation, in the endeavor thus to modify the subject's excessive cellular plethora. Spontaneous hemorrhage is sometimes followed by improvement, for obvious reasons.

Temporary relief, with a definite diminution in the size of the spleen and modification of the hemoglobin and erythrocyte estimates, has been reported as the result of systematic radiation with the *x*-ray, by the technic used in the treatment of leukemia (*q.v.*).

Of the numerous drugs recommended for the relief of the symptom-complex, the iodids (Hirschfeld<sup>58</sup>), and quinin and iodid of mercury ointment (Begg and Bullmore<sup>59</sup>) at least merit mention. Oxygen inhalations have been attended by equivocal results, and the same is true of arsenic, sodium nitrate, and thyroid substance, all of which have been used with the hope of a cure. Formerly splenectomy was performed (Schneider<sup>60</sup>, Axel<sup>61</sup>), but on irrational grounds, for the operation has invariably proved fatal, and is no longer advised.

A dry, virtually iron-free diet, with no alcohol, and a minimum of spices, condiments, tobacco, tea, and coffee is helpful, and drugs of the chalybeate, coal-tar, and vasodilator groups should be absolutely proscribed.

#### BIBLIOGRAPHY.

1. "Chlorosis," London, 1897.
2. *Medizin. Klinik.*, 1915, x, 80.
3. *Brit. Med. Jour.*, 1895, i, 881; ii, 1473.
4. Albutt and Rolleston, "System of Medicine," N. Y., 1909, v, 704.
5. Da Costa: "Clinical Hematology," Phila., 1903, 279.
6. *Berl. klin. Woch.*, 1913, C, 2409.
7. *Jour. Med. Research*, 1916, xxxiv, 469.

8. Practitioner, 1888, xci, 81; Lancet, 1903, i, 283, *et seq.*; Brit. Med. Jour., 1907, ii, 1299.
9. Faust and Tallquist: Archiv. J. exper. Path. n. Pharmak., 1912, lvii, 375.
10. Sosaki: Biochem. Zeitschr., 1914, lix, 429.
11. Aederberg: Berl. klin. Woch., 1914, li, 585.
12. Seyderhelm: Tr. Deutsch. path. Gesellschaft., 1914, xvii, 456.
13. Bull. Johns Hopkins Hosp., 1913, xxiv, 322.
14. Brit. Med. Jour., 1915, i, 407.
15. Archiv. Int. Med., 1913, xii, 6.
16. Esch. Deutsch. med. Woch., 1911, xxxvii, 1943; Wien. klin. Woch., 1913, i, 16.
17. Münch. med. Woch., 1913, 6, 17.
18. Policlinico, 1915, v, 9.
19. Russk. Vrach., 1914, vi, 27.
20. Klemperer: Therap. d. Gegenio., 1913, liv, 385.
21. Lee, Minot, and Vincent: Jour. Amer. Med. Assn., 1916, lxvii, 719.
22. Decastello: Deutsch. med. Woch., 1914, xl, 639.
23. Journ. Amer. Med. Assn., 1916, lxvii, 721.
24. Berlin klin. Woch., 1913, L, 1509.
25. Amer. Jour. Med. Sc., 1914, clviii, 147.
26. Berl. klin. Woch., 1912, lxix, 1357.
27. Wien. klin. Woch., 1912, xxv, 1311.
28. Jour. Amer. Med. Assn., 1913, lvi, 1913.
29. Wien. klin. Woch., 1914, xxvii, 1141.
30. Boston Med. and Surg. Jour., 1908, clviii, 183.
31. St. Louis Med. Rev., 1909, lviii, 113.
32. Amer. Jour. Med. Sc., 1904, cxxvii, 563.
33. Jour. Exper. Med., 1907, ix, 51.
34. Cited by Grawitz: Verhandl. d. Berl. Med. Gesellschaft., 1909, xxxix, 162.
35. Lancet, 1906, ii, 1654.
36. Cited by Grawitz: *Loc. cit.*
37. Modern Clinical Medicine, N. Y., 1906, 368.
38. Texas State Jour. Med., 1916, xi, 529.
39. Am. Surg. Jour., 1916, lxiii, 35.
40. *Ibid.*
41. Amer. Jour. Med. Sc., 1900, cxix, 54; *Ibid.*, 1902, cxxiv, 781.
42. Ann. Surg., 1915, xlviii, 315.
43. Ann. Surg., 1913, lxii, 158; *Ibid.*, 1915, cxiv, 172.
44. Jour. Amer. Med. Assn., lxvi, 716.
45. Wien. klin. Woch., 1889, ii, 435.
46. Arch. Ital. di pediat., 1890, viii, 175.
47. "Pediatrics," Phila., 1901, Ed. 3, 889.
48. Ann. Surg., 1915, xlviii, 679.
49. Compt. rend. Acad. d. sc., 1896, cxxiii, 894.
50. Jour. Med. Research, 1916, xxxiii, 323.
51. Jour. Amer. Med. Assn., 1916, lxvi, 20.



52. Arch. Int. Med., 1914, xiii, 76.
53. *Ibid.*, 1916, xvii, 582.
54. Arch. Int. Med., 1916, xvii, 203.
55. Allbutt's "System of Medicine," N. Y., 1909, v, 937.
56. Bull. Soc. Med. des Hop., Paris, 1899, xvi, 579.
57. Amer. Jour. Med. Sc., 1903, cxxiv, 187.
58. Berl. klin. Woch., 1907, xlv, 1302.
59. Edinburgh Med. Jour., 1905, xvii, 481.
60. Wien. klin. Woch., 1907, xx, 413, *et seq.*
61. Folia Hematolog., 1905, ii, 685.
62. Jour. Lab. and Clin. Med., 1917, ii, 552.
63. Journal of the American Medical Association, 1917, lxi, 1919.
64. St. Paul Med. Jour., 1917, xix, 43.
65. Von Noorden: Merck's Reports, 1917, xxviii, 422.
66. Journ. Amer. Med. Assoc., 1917, lxviii, 747.

# Diseases of the Ductless Glands

BY

CHARLES E. DE M. SAJOUS, M.D., LL.D., Sc.D.,

Fellow of the College of Physicians and of the American Philosophical Society; Professor of Therapeutics, Temple University of Philadelphia, Department of Medicine, etc.;

AND

LOUIS T. DE M. SAJOUS, B.S., M.D.,

Associate Professor of Experimental Therapeutics, Temple University of Philadelphia, Department of Medicine; Instructor in Laryngology, Graduate Medical School of the University of Pennsylvania.



# Diseases of the Ductless Glands.

---

## FOREWORD.

To treat intelligently any of the diseases of this class, it is necessary to understand with some degree of accuracy the functions of the organs affected. The authors deemed it advisable, therefore, to precede the disorders of each organ with a summary of its physiology. Inasmuch, however, as this phase of our knowledge is still shrouded in considerable obscurity, the more salient conceptions are alone presented, selecting those which seemed most clearly to explain the clinical phenomena observed. The views of the senior author are introduced without special emphasis among the many submitted, the purpose being to afford the reader a general review of the subject in its bearing upon the diseases to which the ductless glands are liable. Special stress has been laid, however, upon the treatment of these diseases.

## DISEASES OF THE ADRENAL GLANDS.

**General Considerations.** Each adrenal is constituted of two portions—the external or cortex, and the internal or medulla. The former, the cells of which contain lipoid or fat-like substances, is derived from the same mesoblastic tissues as the primitive kidney, while the medulla yields the “chromaffin reaction,” and is derived from the sympathetic system.

The cells of the medulla, disposed, in man, in columnar masses, are generally separated from the lumina of capillaries or small veins only by an endothelial membrane and contain granules soluble in water and in alcohol, but insoluble in ether or xylol, which embody the characteristic property of chromaffinity and impart a brown coloration to the cut surface as a whole when fixed in formalin containing a salt of chromic acid. According to Stoerk and von Haberer,<sup>1</sup> the chromaffin substance develops in the form of intracellular granules, which,



when sufficiently dense, diffuse out of the cells into the adjoining small vessels, and appear in the adrenal venules as a yellowish brown, refractile, mucoid material. This material, held to constitute the adrenal secretion, passes into the vena cava, and thence into the general circulation.

The adrenal cortex consists essentially of columnar epithelial cells disposed variously in spherical or oval groups, parallel columns, or thin, anastomosing cords and separated by thin partitions of connective tissue. As in the medulla, the parenchymal cells are disposed in close apposition to blood-vessels. A special characteristic is their wealth in lipoid granules, believed chiefly to be made up of cholesterin esters. As in the cells of the medulla, there are also contained in the cortical cells, less abundantly than the main product, other granulations—some pigment granules—of unknown significance.

Both portions of the adrenals receive an abundant blood-supply, brought to them through three sets of arteries. The medulla receives not only blood which has circulated through the cortex from the capsule of the organ, but also blood reaching it through the perforating arteries of Srdinko,<sup>2</sup> which travel to the medulla from the capsule without dividing. Likewise abundant is the nerve supply of the adrenals, especially in their medullary portions. Many nerve-bundles, which pass through the capsule and cortex, reach the chromaffin tissues, around the cells of which they form actual arborizations. Plexuses of non-medullated and some medullated fibers, connected with sympathetic ganglion cells, occur in the capsule of the gland, while in the medullary and even the cortical tissues themselves are also to be found interconnected groups of sympathetic ganglion cells.

The adrenal medullæ embody the greater part of the chromaffin tissue existing in the organs, but there are also subsidiary chromaffin structures, viz., the carotid glands, the tympanic glands, the parasympathetic organ of Zuckerkandl—the latter located anteriorly to the bifurcation of the abdominal aorta—and the chromaffin inclusions in the sympathetic ganglia. Subsidiary interrenal tissues, or “adrenal rests,” corresponding in cellular structure to the adrenal cortex, and possessing no chromaffin property, likewise exist in nearly all

instances; they occur either in or around the adrenals themselves or the kidneys, in the vicinity of sympathetic nerve plexuses, in or near the liver or pancreas, at various points in the retroperitoneal space, and in relation to the reproductive organs, *e.g.*, along the spermatic cord, between the testicles and epididymes, in the testicles, or in the ovaries or broad ligament. These subsidiary adrenal tissues are capable of undergoing a considerable degree of compensatory hypertrophy where the main repository of adrenal tissues—the adrenals themselves—has been gradually destroyed or removed.

Functionally, the two portions of the adrenals appear to play different rôles. Our information as to their physiological activities is based chiefly upon extirpation experiments, and upon studies of the effects of adrenal extracts or principles. Whereas, upon removal of a single adrenal little inconvenience, as a rule, results, excision of both adrenals in animals regularly causes death within thirty-six hours. Rapidly developing disease of both adrenals in man likewise causes early death, and sudden destruction of a single adrenal, as by hemorrhage, may cause death where the opposite organ is already deeply impaired through disease of its vascular or nervous supply.

The nature of the (eventually fatal) effects of adrenal destruction upon the system as a whole is suggested by various facts. Bilateral adrenalectomy is followed, in the first place, by a marked fall of blood-pressure, and a feeble, frequent heart action. Injections of the adrenal principle, adrenalin or epinephrin, on the other hand, induce a characteristic rise of the blood-pressure and strengthening of the heart beat, due to direct and powerful excitation of the contractile vessel walls and of the cardiac muscle. These observations suggest that the morbid phenomena attending adrenalectomy may be due, at least in part, to removal of a normal supporting influence exerted upon the motor functions of the circulatory tract, presumably by the adrenal secretion poured into the blood-stream. The marked similarity of the effects of adrenalin on various structures of the body to those of stimulation of the sympathetic nervous system, whether electrically or by some other means, has also led to the view that the essential rôle of the adrenals is to afford a supporting or tonifying influence to the entire group of functions under control of this system.

According to Sajous, Sr.,<sup>3</sup> the adrenal secretion influences respiration and general metabolism. After passing into the blood, it normally becomes an essential constituent of the hemoglobin, absorbs oxygen from the air in the lungs, and subsequently, by yielding it to the peripheral cellular structures, sustains general tissue respiration. Bernstein and Falta,<sup>4</sup> indeed, have in fact noticed that injections of adrenalin caused an increased consumption of oxygen as well as an increased carbon dioxide excretion. That an excess of adrenal secretion, brought about by the injection of adrenalin, tends to induce a rise in the body temperature is, moreover, a well-known fact; conversely, adrenal insufficiency leads to hypothermia.

An *antitoxic function* on the part of the adrenals has been suspected by Abelous and Langlois,<sup>5</sup> the glands being presumed normally to destroy poisonous products of muscular activity as well as toxic materials of bacterial origin. Sajous, Sr., however, holds that the adrenal secretion carries on this antitoxic function only in conjunction with the products of other ductless glands, the pancreas and thyroparathyroid apparatus in particular. According to D. E. Jackson,<sup>6</sup> one of the functions of the adrenals is to assist, by means of their internal secretion, in counteracting pathological processes or products which tend to cause abnormal constriction of the bronchioles—a view which coincides with those of the writers previously mentioned.

As regards the influence of the adrenal secretion on the blood-pressure, Stewart and Rogoff<sup>7</sup> have ascertained that there occurs a definite rate of spontaneous liberation of adrenin (epinephrin) in cats, viz., 0.0003 milligram to 0.001 milligram a minute for each kilogram of animal; yet the general trend of recent experimental work has been against the conclusion that, physiologically, epinephrin acts *constantly* as an augmentor of blood-pressure. Hoskins and McPeck<sup>8</sup> observed upon practising various degrees of massage over the adrenals, thus liberating varying doses of adrenin in the circulation, a distinct depression from small discharges, but a considerable rise of pressure when the glands were vigorously manipulated. The same experimenters found, however, that where the initial blood-pressure was very low—40 millimeters Hg.—only purely pressor effects could be obtained. This would indicate

that the excretion of adrenin is in at least one sense a sustaining influence on vascular tension, serving to increase the latter when it tends to descend below normal. This view of the adrenals as an emergency organ corresponds with the well-known findings of W. B. Cannon in cats, that under the influence of emotions such as rage or fear, or of great excitement, an excess of adrenin is automatically discharged into the bloodstream, the results being an increased liberation of sugar from the liver, a prompt abolition of fatigue, increased coagulating power of the blood, and an improved flow of blood to the heart, lungs, central nervous system, and skeletal muscles, the digestive processes being meanwhile temporarily inhibited. That the increased adrenal function is evoked through nervous action is indicated by the experimental observation of Mackenzie<sup>9</sup> that nervous stimuli, especially of the sympathetic—piqure or splanchnic excitation—cause an increased secretion of adrenin. According to Sajous, Sr.,<sup>10</sup> the pituitary body is directly connected with the adrenals by nerve-paths, and may thus itself influence the activity of these organs. J. F. Gaskell,<sup>11</sup> noting the common embryologic origin of the sympathetic nervous system and the chromaffin cells, expresses the view that regulation of the vascular tree in general is effected in a twofold manner, viz., both by the sympathetic nerves (vasoconstrictor fibers) and by the secretion of adrenin.

Confirming the assertion of Sajous, Sr., many years ago,<sup>12</sup> that certain drugs produce tonic effects on the heart through the adrenals rather than by direct stimulation of the heart muscle, Gley<sup>13</sup> has recently reported effects from such violent cardiac poisons as anagyrin and nicotin which he could ascribe only to the adrenals, and has been led to recognize the existence of a class of heart stimulants which act through these organs. Finally, Cannon and Cattell<sup>14</sup> have observed that adrenalin in a small dose is capable of markedly increasing the activity of the thyroid gland; the same result was observed when the splanchnic nerves to the adrenals were stimulated. A strong probability is thus suggested that, besides directly favoring oxidation through secretion of epinephrin—the previously mentioned conclusion of Sajous, Sr.—the adrenals, when stimulated, increase general metabolism through excitation of the thyroid.



In conclusion, it should be mentioned that, according to some, the removal of the adrenal cortices in bilateral adrenalectomy plays a much more important part in the succeeding fatal termination than has hitherto been believed. Voegtlin and Macht<sup>15</sup> detected in the adrenal cortex a body having a digitalis-like action, and Iscovesco<sup>16</sup> found in it a cardiotonic lipoid. That the adrenal cortex is capable of exerting a marked stimulating influence on the essential organs of reproduction has long been recognized.

A probable influence of the adrenals upon growth in general has been experimentally noticed by F. de Mira,<sup>17</sup> who, upon removal of the left adrenal in the young cat and dog, found the growing animals smaller and thinner than controls from the same litters; the bones in particular were notably lighter in the operated animals.

### ADRENAL INSUFFICIENCY (HYPOADRENIA).

Cases of insufficiency of the adrenal functions (hypoadrenia; hypoadrenalism) may conveniently be classified into three forms: (1) Functional hypoadrenia, in which the adrenals, while not organically diseased, are functionally impaired through hypoplasia, or because of debilitating influences, such as fatigue, starvation, etc., or old age. (2) Progressive hypoadrenia, or Addison's disease, in which the functions of the adrenals, either directly or through their secretory nerves, become progressively reduced through organic lesions, such as tuberculosis, cancer, fibrosis, etc. (3) Terminal hypoadrenia, a more or less tardy complication of infectious diseases and toxemias, due to exhaustion of the secretory power of the adrenals from previous hyperactivity.

**Functional Hypoadrenia.** The symptoms of this condition are such as would be expected, in view of the physiological properties of the adrenals already mentioned, to result from interference with the activity of these organs. They consist chiefly of general motor asthenia, a tendency to hypothermia, with sensitiveness to cold and actual coldness of the extremities, low blood-pressure, weak heart-action and pulse, anorexia, psychasthenia, anemia, and slow metabolism.



In infancy and childhood the effects of hypoadrenia due to tardy development of the adrenals become manifest particularly after the period of transference of immunizing substances from mother to offspring with the maternal milk has terminated through weaning. A persisting hypoplasia of the infantile adrenals, thus rendered unable properly to assume the burden of adequate tissue oxidation and defence against infection, is apt to result in pallor, a pasty appearance, or emaciation, cold hands and feet, flabbiness of the muscles, a deficient or capricious appetite, and an unusual susceptibility to infections of all types.

In the adult, inherently weak adrenals lead similarly to a feeble circulation, to a tendency to adiposis, and probably to the appearance of bronze spots. More frequently, however, the condition is an acquired adrenal weakness due to exhaustion of these organs through excessive secretory activity. A frequent cause of temporary adrenal insufficiency is physical fatigue. In the war in Europe, many instances of otherwise unaccountable physical depression, with a tendency to hypothermia, following repeated arduous military tasks, have been definitely ascribed to adrenal exhaustion, the demands upon these organs as regards general oxidation and (according to the view of Abelous and Langlois,<sup>18</sup> in overcoming the unusually abundant toxic products of muscular activity), having temporarily overwhelmed their functioning power. In a number of instances Sergeant's white line phenomenon—generally considered indicative of adrenalin deficiency—was noted by Merklen<sup>19</sup> in the presence of hypothermia and general physical misery in soldiers. Carl<sup>20</sup> observed absence of the chromaffin reaction in the adrenals of a bicyclist who had succumbed from extreme exertion, as well as in those of frogs after strychnin convulsions. Other possible causes of temporary adrenal insufficiency are deficient food, and excessive venery and masturbation. Sézary<sup>21</sup> recognizes, applying to it the term "hypopinephry," a permanent incapacity of the adrenals to protect the body against infection. Experimental work by F. C. Mann<sup>22</sup> has tended to indicate that adrenal insufficiency may contribute or lead to the production of peptic ulcers, a large percentage of dogs and cats dying after adrenalectomy showing such ulcers at points of hemorrhage in the gastric mucosa,

In old age impairment of the adrenal functions is a result of the retrogressive changes frequently taking place in these glands in later adult life. Landau<sup>23</sup> found the adrenals markedly shrunk and hypovascular in aged subjects, while others have found distinct fibrosis, especially in the medullary portion, in old experimental animals. The asthenia of old age thus finds a normal explanation in the attendant defective supply of adrenal secretion, and the possibility is even to be thought of that integrity of the adrenals plays an important part in the promotion of longevity.

*Prophylaxis and Treatment.* Much is possible in the way of remedial correction in each of the three types of functional hypoadrenia mentioned. In the infantile cases, distinct prophylactic possibilities are presented, the aim being by all means to obviate such early injury to the adrenals and other protective organs by infection or intoxication as would leave them in a permanent state of functional impairment. Especially to be emphasized in this connection is the immunizing value of maternal milk, the lack of which, so frequent under prevailing conditions of rather indiscriminate substitutional feeding, places an unnatural strain upon the autoprotective functions for which they are at the time practically unprepared.

Idiopathic hypoplasia of the adrenal glands is doubtless to be guarded against largely in the same manner, all measures being taken to secure as perfect a nutritive condition of the infant as possible. Of these measures, proper assimilation of good maternal milk is obviously the most essential, and where the milk of the child's own mother is at fault, that of a healthy wet-nurse is to be sought in preference to any other form of substitution.

In children past the period of nursing, the prophylaxis of hypoadrenia consists again in avoiding excessive strain on the adrenals through infection and intoxication. The use of milk free from dangerous bacterial contamination continues, for a time, to be one of the essential measures. Proper hygiene in the school, as well as at home, are also necessary features, and the advisability of preventing at any time a severe and continuous absorptive infection, as from the mouth or pharynx, is likewise to be borne in mind.

In the actual presence of functional hypoadrenia, various remedies, other than adrenal gland itself, are available. In feeble children ten or twelve years of age, a capsule containing 1 grain (0.06 Gm.) each of dried thyroid and Bland's mass, with 2 grains (0.12 Gm.) of dried suprarenal, given three times daily, will often prove very beneficial, especially if properly supported by hygienic measures. D'Ælsnitz<sup>24</sup> has confirmed the utility of dried adrenal gland in the presence of moderate hypoadrenia in childhood, manifested in retardation of growth and of walking, emaciation, flabbiness of the muscles, fatigue, physical and mental indolence, and low blood-pressure. The frequent efficacy of small doses of mercury, in the form of the biniodide, or of calomel, as a stimulant to the functions protecting the system against infection, including that of the adrenals, is also worth remembering. Meats, and especially milk, are of marked value in the diet in these cases, and where the circulation is weak, or the nervous reflex functions as a whole apparently inactive, small doses of digitalis or of strychnin are likely to prove of service.

The functional hypoadrenia of adults is frequently susceptible of improvement or correction through the removal of influences placing an undue functional burden on the adrenals. In the temporary adrenal exhaustion of soldiers, complete physical rest for as long a period as is necessary to permit the organs to recuperate is obviously paramount. The strain on the adrenals having often been relatively brief, no obstacle may exist to an eventual complete recovery of functional power. A significant fact in these cases, however, is the probable harmful effect of repeated fatigue in the presence of already overburdened adrenals. Thus, Morat and Doyon<sup>25</sup> refer to the aggravating influence, at times even resulting in sudden death, observed upon instituting experimental fatigue in animals deprived of their adrenals. Furthermore, not only is the physical incapacity of soldiers with exhausted adrenals a feature requiring attention, but their powers of resisting infection are also to be remembered as being unfavorably influenced by the adrenal impairment. To stimulate actively the functions of fatigued adrenals by drugs would obviously tend to ultimately aggravate the hypoadrenia, possibly after a brief period of improvement. The use of dried suprarenal or of adrenalin,

however, to make good temporarily the lack of adrenal product or products in the system is not open to this objection, and has actually proven of considerable remedial value, both in accelerating recovery from the hypoadrenic depression and in protecting the individual from complicating disorders such as infection. Josué<sup>26</sup> notes that, to obtain the best results, generous dosage of either the pure principle or the dried gland is required. He has found adrenalin by the mouth efficient if used to the amount of 1 to 4 or even 5 mils (15 to 80 *m.*) of the 1:1000 solution in a day, in divided doses. Three mils could thus be given daily for a month or more without harm. In the hypodermic use of the drug he recommends a "slow absorption" method, carried out by injecting under the skin 250 to 500 mils ( $\frac{1}{2}$  to 1 pint) of normal saline solution to which 1 mil (15 *m.*) of adrenalin solution and 0.01 gram ( $\frac{1}{100}$  gr.) of novocain have just been added. The dosage is 0.5 to 2 mils (8 to 30 *m.*) a day, 0.5 mil (8 *m.*) being preferably not exceeded as the single dose. Extracts of the whole adrenal, given by mouth in daily amounts of 0.2 to 0.4 gram (3 to 6 gr.), in two doses, or hypodermically in a daily amount of 0.1 gram ( $1\frac{1}{2}$  gr.), in some cases even proved distinctly superior to adrenalin.

Attention has been called by Tom A. Williams<sup>27</sup> to the similarity of many of the manifestations of neurasthenia to those of hypoadrenia. Where, in a neurasthenic, subnormal blood-pressure and temperature, together with pigmentary skin changes, are noted, hypoadrenia should, according to this author, be suspected as an underlying cause. Treatment directed toward overcoming this condition might be expected to be particularly helpful in these cases.

In the hypoadrenia of old age, as in that due to overburdening of the adrenals at any period of life, active stimulation of the adrenals by drugs is unwise, not only on account of the possibility of accelerating the retrogressive changes in these organs through excessive functional activity, but also, in the former group of cases, owing to the frequent coexistence of arteriosclerosis. Other measures, however, can be utilized to help make up for the impaired adrenal function. In the diet, milk, a product in which the adrenal principle is present, as shown by Sajous, Sr., affords a ready means of compensation,

the antitoxic properties of milk, for example, tending to replace those of the lacking adrenal product. Another beneficial dietetic measure is the daily ingestion, in addition to simple, though varied, food, of the expressed juice (uncooked) of 1 pound of fresh beef, taken alone or in a soup. A hygienic mode of living, with exercise out-of-doors in reasonable amount, and marked caution in sexual matters—overindulgence proving very debilitating in such subjects—is also important. In the presence of marked general asthenia, administration of adrenal products may be necessary. Thus, in the case of a lady of 72 years, confined to bed through sheer weakness and listlessness, and observed by Horton,<sup>28</sup> adrenalin yielded striking results, though strong tonics, including strychnin, had failed. The output of urine, previously reduced to 10 or 12 ounces a day, rose in two days to 40 ounces, and in a few days the patient was up. Continued use of the adrenalin proved essential, the previous debility returning whenever the drug was temporarily left off. Of interest in relation to this case is the experimental observation by Marshall and Davis<sup>29</sup> of a marked lowering of renal efficiency in adrenalectomized cats, even where the blood-pressure had not yet sunk below normal.

**Terminal Hypoadrenia.** This term has been applied by Sajous, Sr., to the form of adrenal insufficiency which occurs late in the course of an acute febrile disease as a result of exhausting functional overactivity. The condition of the adrenals may have been aggravated through temporary local lesions induced in them during the course of the general disease. Pathologically, adrenals thus affected differ, in the majority of instances, from those of functional hypoadrenia in being not only enlarged and hyperemic, sometimes with small areas of hemorrhage, but also the seat of a passive type of congestion due to loss of resiliency of their sinus-like vessels, with consequent impediment to the circulation through the organs. Suppuration in the adrenals is at times also a complication in the presence of a strepto-, pneumo-, or meningococcic infection in other structures.

The exhaustion of the adrenal function in the presence of infections probably arises from the fact that these organs are concerned in the protection of the organism against intoxica-



tions and bacterial invasions, and are therefore apt to be functionally called upon to a marked extent in the presence of disease. Comessatti<sup>30</sup> noted that in diseases of long duration the epinephrin content of the adrenals was far less than in cases of sudden death. In typhoid fever and diphtheria, hypoadrenia is observed with special frequency. Other conditions in which it has been noticed by various authors include lobar and bronchopneumonia, septicemia, scarlatina, measles, mumps, erysipelas, acute nephritis, certain forms of tonsillitis, etc. Fulminating or malignant types of the acute infections are frequently to be explained on the basis of adrenal insufficiency, though more commonly the hypoadrenia is witnessed as a late complication after a period of gradual exhaustion of the adrenals from continued toxemia has elapsed. The manifestations of the condition are similar to those mentioned under Functional Hypoadrenia, viz., unusual asthenia, circulatory weakness, a relatively low temperature, vascular hypotension, chilliness, pallor and Sergeant's white line phenomenon, together with anorexia, nausea, vomiting, diarrhea, a tendency to fainting spells or sudden exitus from cardiac failure, and a liability to septic complications or relapse of the existing disease.

Tscheboksaroff,<sup>31</sup> tracing experimentally the secretory activity of the adrenals in animals poisoned with diphtheria toxin, noted, as we might expect, at first an increase of epinephrin in the blood, followed by a gradual decrease and total disappearance. From the pathological standpoint, Moltschanow<sup>32</sup> found the adrenals regularly the seat of lesions in 42 children succumbing to infectious diseases, including 29 cases of diphtheria. Beresnegowski<sup>33</sup> holds acute peritonitis pre-eminent among infections in the production of adrenal lesions, and found marked histological lesions of the adrenal cortex in 50 per cent. of cases examined *post-mortem*. Remlinger and Dumas,<sup>34</sup> observing sudden hypothermia and heart weakness in 4 out of 100 soldiers suffering from bacillary dysentery, found proof at the autopsies that the adrenals had been the cause of these symptoms. Sergeant<sup>35</sup> has noted hypoadrenia in soldiers as a result of traumatic shock, typhus fever, choleric form diarrhea, Asiatic cholera, and chloroform anesthesia, as well as of physical exhaustion.

*Treatment.* Marked benefit has been noted clinically from adrenal products in cases presenting the symptoms above referred to. In emergency cases, administration of the pure principle epinephrin is advisable. The observations of Josué (v. ante under Treatment of Functional Hypoadrenia) and others, have established the efficacy of the subcutaneous and oral routes of administration, though where there is immediate danger, the intravenous route, the epinephrin being given in normal saline solution, will procure even more prompt results. Moizard<sup>36</sup> recommends use of the pure principle as soon as asthenia and low blood-pressure occur in any infection, and Kirchheim,<sup>37</sup> among others, found 0.6 to 1.5 mils (10 to 24 *m.*) doses of the 1:1000 solution safe when given hypodermically in the collapse of pneumonia, diphtheria, and scarlet fever. Personal observations as to the benefit from adrenalin in adynamic cases of typhoid fever and other acute infections have been quite convincing. In cardiovascular failure in pneumonia and typhoid and paratyphoid fevers, Mansvetova<sup>38</sup> found 0.5 mil (8 *m.*), hypodermically every hour or hour and a half, the best dose for persistently improving the blood-pressure. In hypoadrenia due to traumatic shock, infections, exhaustion, etc., Sergeant<sup>39</sup> thinks it best to combine oral with hypodermic use, administering 2 to 3 milligrams ( $\frac{1}{30}$  to  $\frac{1}{20}$  gr.) of the pure principle in 4 to 6 doses hypodermically and 1 to 2 milligrams ( $\frac{1}{60}$  to  $\frac{1}{30}$  gr.) orally.

In cases in which the advent of asthenia and low blood-pressure is gradual, dried suprarenals (*Suprarenalum siccum*, U. S. P.) may appropriately be given by mouth in doses of 0.1 to 0.3 gram ( $1\frac{1}{2}$  to 5 gr.) in capsules three times a day. According to Josué,<sup>40</sup> hypodermic use of a suitable preparation of the whole gland in a daily dosage of 0.1 gram ( $1\frac{1}{2}$  gr.) gives good results.

The combined use of adrenal and pituitary preparations has been specially urged in the circulatory weakness and adynamia of infectious diseases by Kepinow and others. Rohmer,<sup>41</sup> in the hypoadrenia of pneumonia, diphtheria, and typhoid fever in children, claims to have found the combined intravenous administration of pituitrin, 0.25 mil (4 *m.*), and adrenalin solution 0.5 mil (8 *m.*), in young children, with doses twice as large in older children, superior to other circulatory stimulants.

After cardiac adynamia has passed off under the influence of adrenal therapy, dried thyroid (*Thyroideum siccum*, U. S. P.), 0.03 gram ( $\frac{1}{2}$  gr.), together with strychnin, 0.001 gram ( $\frac{1}{60}$  gr.), and Bland's pill-mass, 0.06 gram (1 gr.), may be added to the above-mentioned capsules of dried adrenal gland, to accelerate convalescence.

**Chronic Progressive Hypoadrenia, or Addison's Disease.**

The symptoms of this disorder, generally due to chronic lesions, often tuberculous and occasionally cancerous, of the adrenals, are in the main those of functional hypoadrenia—already summarized—with certain special features superadded. In some cases the adrenal insufficiency is not due primarily to disease of the adrenal tissues themselves, but to lesions in, or in the vicinity of, their secretory nerves, whether in the ganglia of the suprarenal plexuses, the splanchnic nerve-trunks, or the spinal cord near the point of emergence of the adrenal pathways. Pressure on such nervous pathways can doubtless result finally in interference with the adrenal functions, either through interruption of secretory nerve impulses or through primary excitation followed by lasting exhaustion of the glandular parenchyma. In cases recorded by Semmola and Brault, for example, bronzing occurred through protracted pressure upon the semilunar ganglia and solar plexus.

Lowered temperature, progressive asthenia, weak heart-action, low blood-pressure, and dyspnea are manifest results of the increasing hypoadrenia. Emaciation through deficient anabolism, anorexia through lowered demand of the tissues for their pabulum, vomiting from gastroparesis and deficient evacuatory peristalsis, and constipation through intestinal atony or diarrhea from passive congestion of the mucosa, are all subsidiary effects of the underlying adrenal impairment. Sergeant's "white line," a broad, white streak which gradually appears upon drawing a finger pulp over the abdominal skin, and passing away after three or four minutes, seems to be in some degree a diagnostic sign. The general impairment of muscular tone and consequent asthenia are probably, in part at least, due to imperfect carbohydrate metabolism, Mackenzie<sup>42</sup> having found a diminution of the power to form glycogen from glucose in adrenalectomized dogs; sugar administered to such animals was neither oxidized nor stored as glyco-

gen, but was entirely eliminated as such in the urine. The effect of a general lack of glycogen on motor functions is obvious.

The bronzing of Addison's disease is not an essential symptom of the condition, some cases, in fact, dying before the pigmentation has appeared. As clinical observations and animal experiments have shown, it is an indication, where present, of *advanced* lesions in the adrenals or of their nerve-supply. As a rule, it occurs only in the course of a *gradual* impairment of the adrenals. Brown-Séquard, indeed, having already noted many years ago that it appeared particularly in animals in which operations on the adrenals were so performed as to cause death only after some months. Lippmann,<sup>43</sup> however, has reported a case of acute Addison's disease in a previously healthy young sailor, in which low blood-pressure, intense asthenia, signs of intoxication, and *pigmentation* followed in rapid succession, with death on the eighteenth day.

Other common manifestations of advanced Addison's disease include a tendency to syncope and to impairment of vision and hearing, due to ischemia of the brain and special sense structures, and a toxemia, due to deficient antitoxic function, finding its symptomatic expression in headache, irritability, muscular twitchings, delirium and convulsions. Death may occur from progressive asthenia, adrenal apoplexy or interstitial hemorrhage due to excessive congestion of these organs when the seat of advanced lesions, or from intercurrent disease.

*Treatment.* The most logical procedure in chronic disease of the adrenal tissues causing impaired function is obviously to replace the missing glandular parenchyma through adrenal grafting. The numerous attempts made at experimental grafting in adrenalectomized animals have been, as a rule, disappointing, though Busch, Leonard, and Wright<sup>44</sup> have succeeded in transplanting the adrenal of a rabbit into the kidney of another rabbit previously subjected to unilateral adrenalectomy, and upon removal of the remaining adrenal after thirty-six days, witnessed recovery of the animal, indicating functional activity on the part of the transplanted organ. Cases of transplantation of an animal adrenal into a human subject suffering from Addison's disease have often resulted dis-



astrously, however, death sometimes following within one or a few days. A reasonable explanation of these unfortunate results is, however, not hard to find. When one calls to mind that less than one-tenth of the average total amount of adrenal tissue with which the organism is provided has been experimentally shown to be sufficient to satisfy completely the demands on adrenal function under ordinary conditions, and that upon gradual pathologic destruction of the adrenal glands such readjustments of function may occur in other ductless glands as will enable the organism to live, for a time at least, with even less than one-tenth of the normal amount of adrenal tissue still functioning, it is obvious that the addition of an excessive amount of adrenal tissue to the system may result in so inordinate an adrenal functional effect as will dangerously disturb the body as a whole. Such a condition seems to have been responsible for at least some of the deaths promptly following clinical adrenal transplantation, Courmont,<sup>45</sup> for example, having noticed after the procedure a "formidable hyperthermia" which persisted until death in collapse in spite of the absence of all signs of infection. Evidently, in grafting, it is eminently necessary to adjust the size of the graft to the actual needs of the recipient, remembering that the more advanced the preceding adrenal destruction the greater is likely to be the harmful effect of a large addition of adrenal tissue. The occurrence and degree of such an effect may, of course, be expected to depend upon, and vary according to, the extent of absorption of the adrenal product from the new adrenal, as well as the rapidity with which a blood-supply permitting of functional activity in the graft is developed.

In what appears to be a successful case of adrenal grafting reported by D. M. Morton,<sup>46</sup> transplantation of an adrenal from a patient dead of heart disease was effected into the lower portions of the recti abdominis muscles in a woman of 35 with typical Addison's disease, deemed of tuberculous origin. The adrenal transferred was bisected and one-half buried in each rectus. For four days the patient was very ill, but thereafter she improved rapidly, and was soon restored to comparatively good health, with a distinct lessening of pigmentation and rise of blood-pressure, and a gain of 16 pounds in



weight. This case, while sufficiently advanced to show pigmentation, was from the symptomatology not as yet in a critical stage of adrenal deficiency. This may explain the relative lack of an alarming reaction after transplantation of an entire adrenal.

Adrenal transplantation not having so far shown its availability as a routine measure, reliance is usually placed on adrenal opotherapy to make good the deficiency in adrenal function. As Sergeant<sup>47</sup> has stated, Addison's disease can often be benefited, and sometimes even recovered from, through this means. The treatment tends at times, indeed, to induce a compensatory hypertrophy of remaining adrenal tissue which will partially eliminate the deficiency. In 25 of 120 cases collected by E. W. Adams,<sup>48</sup> permanent benefit accrued from such treatment. That careful adjustment of the dosage to the needs of the individual case is essential is illustrated by Bate's case,<sup>49</sup> in which but 0.005 gram ( $\frac{1}{12}$  gr.) of adrenal gland three times a day caused great improvement, with temporary aggravation when the drug was discontinued for a time; while in a case reported by Suckling<sup>50</sup> the daily amount was gradually increased from 0.6 to 12 grams (10 to 175 gr.), likewise with benefit resulting. Failure in the use of the remedy may thus occur either from excess or insufficiency of dosage. Adrenal gland will prove helpful when adrenalin will fail, as shown by Judson Daland.<sup>51</sup>

As guides to the proper dose, records of the temperature and blood-pressure are convenient. Where both are considerably below normal 0.2 gram (3 gr.) of the dried gland (*Suprarenalum siccum*, U. S. P.), twice daily during meals, constitutes an average initial dose, to be increased if necessary, to restore the temperature and blood-pressure to normal. If the dried gland be not available, a glycerin extract of fresh gland may be used, or fresh sheep or ox adrenals given twice a day in doses of 0.3 to 1 gram (5 to 15 gr.). It should be borne in mind, as regards the temperature, that in many instances of Addison's disease, hypothermia is prevented by the febrile reaction attending the underlying disorder itself, *e.g.*, tuberculous infection or carcinoma and the coexisting toxemia.

Appropriate remedies other than dried adrenal include Blaud's mass, to counteract the anemia; glucose, found useful

by Pitres and Gautrelet<sup>52</sup> to lessen the adynamia and sensation of fatigue; creosote carbonate, 0.3 gram (5 gr.) three times daily, in the tuberculous cases, with or without iodids, and mercury, found very effective by Gaucher and Gougerot<sup>53</sup> in a case believed due to syphilitic involvement of the adrenals. Proper treatment of the underlying systemic disorder, where such exists, is, of course, never to be omitted. In a case recorded by Munro,<sup>54</sup> suspected of tuberculous origin, the customary climatic and general hygienic procedures, coupled with courses of tuberculin injections, seemed to restore the patient to good health. Indeed, such cases should always receive treatment addressed to the tuberculous process.

Rest, in or out of bed, according to the stage of the case, is indicated owing to the cardiac weakness and tendency to syncope. The diet should be nutritious but readily digested, milk and meats being especially serviceable. Where motor adynamia of the stomach gives trouble, gastric lavage will give relief. If diarrhea exists, bismuth may appropriately be given.

Pituitary preparations, probably related in their composition and effects to the adrenal products, may prove of some value. Unusual caution is desirable as regards the intravenous administration of blood-pressure-raising remedies, especially the adrenal principle itself, Lowry,<sup>55</sup> for example, having reported a case in which adrenalin intravenously caused general sweating followed by sudden cardiac and respiratory arrest, with recovery only after half an hour's vigorous artificial respiration.

### ADRENAL OVERACTIVITY (HYPERADRENIA).

Considerable experimental evidence is at hand to show that, under the influence of various bacterial toxins, inorganic poisons, and vegetable drugs, pronounced congestion of the adrenals may be produced. Bernard and Bigart,<sup>56</sup> studying the effects of arsenic, mercury, and lead on the adrenal parenchyma, found in the less profound intoxications the histological signs of functional hyperactivity of these organs and, in the more severe intoxications, destructive lesions. W. H. Brown and L. Pearce<sup>57</sup> deem adrenal injury an important fac-

tor in arsenical intoxication, and found that toxic doses of all arsenicals induced adrenal changes, including congestion, hemorrhage, disturbances in the lipoid and chromaffin content, and cellular degenerations and necroses. In the presence of bacterial products, congestion of the adrenals may either be due to the participation of these organs in the defensive process, the accompanying unusual functional activity, in conjunction with a like state in the thyroid gland, or to accumulation in them of blood resulting from high general vascular tension, the latter sometimes leading to focal hemorrhages.

The adrenals, furthermore, are peculiarly subject to hemorrhagic extravasations, apparently through fragility of their richly distributed vascular tree and their proximity to a large arterial trunk, the abdominal aorta, with its relatively high level of blood-pressure. Loeper and Oppenheim,<sup>58</sup> among 150 autopsies taken at random, noted five instances of adrenal hemorrhage visible to the naked eye, and eight more discernible microscopically. In cases in which death had been due to infectious disease, the ratio of adrenal hemorrhages was even much higher. In the newborn, adrenal hemorrhage is exceedingly frequent, 45 per cent. of 250 autopsies having shown this condition.

Both clinical and experimental observations indicate that adrenal congestion occurs as a precursor of adrenal hemorrhage. Hence, the fact that where adrenal congestion due to increased functional demands on these organs exceeds a certain limit, adrenal hemorrhage will result, especially in the presence of such blood-pressure conditions, *e.g.*, a toxic hypertension, as will impose an unusual centrifugal stress on the adrenal vessels.

In the newborn, adrenal hemorrhage may occur within a few moments or days after birth, and entail sudden death. Apparently any condition tending to interfere with proper initiation of the respiratory function predisposes to it. Cyanosis, purpuric spots, convulsions, and death constitute the clinical course of events in such instances. In another group of cases the condition seems due to incapacity on the part of the defensive resources of the infant, perhaps through lack of the immunizing constituents of maternal milk to cope with some endogenous toxemia. The accumulating toxic wastes, causing

a violent elevation of the blood-pressure in adrenals already congested through hyperactivity, break down the resistance of one or more adrenal vessels, and, rupturing them, lead to hemorrhage and such clinical manifestations as abdominal pain, diarrhea, vomiting, and increasing coldness of the extremities, followed by convulsions or cardiac collapse.

In children, a very similar train of events, with death in from a few to forty-eight hours after adrenal hemorrhage, is produced from the action of various infectious conditions, including the exanthemata, diphtheria, septic processes, bronchopneumonia, etc., as well as from ptomaine poisoning, extensive burns, or other severe injuries. Venous stasis and a marked increase of the blood-pressure are, according to Dudgeon,<sup>59</sup> frequently associated conditions. Fever and high blood-pressure in any infection should suggest the possibility of adrenal hemorrhage.

In adults sudden or rapid death from adrenal apoplexy is not as uncommon as is generally believed. Marked abdominal pain, radiating to the back; tympanites, vomiting, prostration, and obstinate diarrhea are the clinical features of this accident. Purpura is far less common than in children, but a yellowish skin discoloration, passing even into the bronzing of Addison's disease, is not infrequently noted. Epileptic seizures, unusual physical efforts, and acute nephritis have been known to bring on adrenal hemorrhage in adults. A common characteristic is the presence of pre-existing adrenal lesions, *e.g.*, tuberculous changes, which predispose the adrenal vessels to rupture when any added strain, such as a rise in blood-pressure due to toxic accumulations, is imposed. Arteriosclerosis involving adrenal vessels has also been found a factor predisposing to hemorrhage. No mention of adrenal hemorrhage being, in general, made in textbooks, the condition has been mistaken clinically for arsenical and other forms of poisoning, cholera morbus, appendicitis, cerebral apoplexy, etc.

#### TREATMENT.

In view of the wide variety of possible causes of hyperadrenia and adrenal hemorrhage, and the consequent multiplicity of symptoms which may precede it, recognition of

the condition before the advent of actual adrenal injury is a difficult matter. High blood-pressure being, however, a frequent determining cause of the hemorrhage, where this condition is observed measures may be taken to diminish, by lowering the blood-pressure, the likelihood of serious adrenal damage. Thus, such drugs as *veratrum viride*, chloral hydrate, nitroglycerin, or even amyl nitrite may be administered, according to indications. Preferable to any of these, perhaps, is saline solution, given by rectum in cases of possible adrenal hemorrhage, subcutaneously in threatening cases; or intravenously in emergency cases. Exerting a detergent action on the blood, and accelerating elimination of toxic materials through the kidneys, saline solution tends indirectly to allay toxic spasm of the vascular system, and will thus effectually assist in obviating excessive stress on the adrenal circulation. Where adrenal hemorrhage has already occurred, evidences of a sudden hypoadrenia are sometimes to be noted. The treatment then applied should be that of terminal hypoadrenia (*q.v.*). If the hemorrhagic focus is not so large and destructive as immediately to endanger life, adrenal or pituitary preparations may prove of marked assistance in tiding the patient over the critical period.

### ADRENAL HEMATOMA.

This condition, sometimes termed "adrenal hemorrhagic pseudocyst," is merely a complication of adrenal hemorrhage, and is generally unilateral. It may cause a fatal termination if rupture of the cyst occurs, its contents entering the peritoneal cavity. The cyst is not of sudden advent, however, but develops gradually, the adrenal tissues becoming destroyed and transformed by it so that its contents comprise not only blood, but cellular detritus, cholesterin crystals, etc., with shreds or remnants of the adrenal cortex lining its walls. While such a cyst may attain a large size, the fact that the opposite adrenal remains uninvolved practically excludes the production of symptoms of altered adrenal function, the signs of the tumor being merely a sensation of weight and pain, due to pressure on surrounding sensitive structures. Subsequently, the patient may suddenly begin to fail, losing weight, and develop-



ing dyspnea, polyuria, hematuria, and even slight bronzing. The termination is usually through rupture into the abdominal cavity.

### TREATMENT.

Excision of the affected adrenal is the procedure of choice, the remaining organ sufficing to carry on the adrenal functions. Either a lumbar or an anterior abdominal incision may be used. Ordinarily, an oblique incision below the last rib is most convenient, or if much space is required, the last rib may be removed. At times the adrenal cyst adheres so tightly to the kidney that the latter has also to be taken out. Küttner<sup>60</sup> has reported a case in which removal of the cyst, adherent to adjacent organs, was facilitated by tapping, a quart of brownish fluid being thus evacuated; recovery followed. Among 11 cases of adrenal cyst found by him in the literature, 5 recovered.

### HYPERNEPHROMA.

This is a special type of tumor presenting microscopically the characteristic features of the adrenal cortex, and developing from bits of adrenal tissue—adrenal rests—either in the adrenals themselves or in the walls of blood-vessels or other structures. They are especially common in the kidney, constituting, according to Albarran and Joubert, 17 per cent. of all renal tumors. Less often they occur in the uterus, ovary, broad ligament, etc., from adrenal rests therein. Benign at first, they gradually exert pressure symptoms in surrounding structures, and later tend to metastasize in the lungs, bones, and brain.

Hypernephroma of the adrenals develops generally between the first and eighth years, and causes a curious form of premature development, as a result of which the child may appear twice or three times its actual age. Observed usually in girls, the condition is characterized by an abundant growth of hair over the face, genitalia, pubis, and sometimes over the entire body, with a swarthy or coppery hue of the skin. The external genitalia often show a marked degree of precocity, the voice is apt to be deep and harsh, muscular strength is unusually well developed, and the body is obese. The effect of the

excess of adrenal tissue is evidently such in these cases as to accelerate growth in general, though the actual gigantism and peculiar facial and other deformities of acromegaly are lacking. According to Glynn and Hewetson,<sup>61</sup> abnormal sex characters do not develop where adrenal hypernephroma develops after the menopause, and are probably absent in adult males.

In hypernephroma of the kidney, hematuria is the most constant and often the first symptom. The bleeding occurs intermittently, but is severe while it lasts. It may precede the development of a palpable tumor by a considerable period. Lumbar pain suggesting lumbago may also at times be the initial symptom; or metastasis in the vertebræ, ribs or other long bones, skull, scapula, etc., or in the lungs, may first indicate the condition. Premature arteriosclerosis and high blood-pressure may be observed, and the skin is typically yellowish or smoky in appearance. According to Gellé, fragments of the tumor may be found in clots passed *per urethram*. Confusion of it with an enlarged spleen is obviated in that it usually occurs on the right side, and is less superficial and movable. It is distinguished from renal calculus in that the pain attending it continues after the hemorrhage. The duration of the disease is from fifteen weeks to eight years, the later stages being marked by emaciation, weakness, secondary anemia, edema from pressure on a vein, delirium, and coma.

Considerable diagnostic value has been attributed by Israel, Neu, and others to the presence of fever during the early stage of hypernephroma, before cachexia has developed.

### TREATMENT.

Since hypernephromas, upon beginning to metastasize or show other signs of malignancy, are likely to progress rapidly, operative exploration of the abdomen is warranted even where their presence is only surmised, viz., where hematuria and an abnormal growth in the abdomen in the kidney region coexist. The mere fact that a hemorrhage into the bladder cannot be accounted for constitutes, according to many, an indication for exploratory incision for hypernephroma. At times a sensation of tension or discomfort experienced by the patient over one kidney, or deep palpation of the two sides, will afford

guidance as to which side should be explored first. In excising the growth, the fatty capsule should also be removed, according to Kusmik, recurrence being otherwise a possibility from malignant infiltration of the fat. By incising in the lumbar region the growth may be removed extraperitoneally.

### TUMORS OF THE ADRENAL BODIES.

**Malignant Tumors.** Malignant hypernephroma, though common in the kidneys, is rare in the adrenals themselves, the chief primary malignant tumors of the adrenals being carcinoma and sarcoma, which occur with approximately equal frequency. The former occurs usually in adult and aged subjects; the latter, in early life. In each, the clinical course is characterized by gradual emaciation and increasing adynamia, with enfeebled cardiac action, anorexia, digestive disturbances, anemia, and occasionally respiratory complications. In the majority of cases, various degrees of the skin pigmentation typical of Addison's disease, ranging from slight yellowness to actual bronzing, are to be noted. According to Israel,<sup>62</sup> two symptoms are of special diagnostic import, signifying adrenal involvement: (1) paroxysms of pain and paresthesias in the absence of a palpable tumor, and (2) a febrile course, noticed by this observer in 57 per cent. of his cases, whereas in renal tumors it was noticed only in 1 to 2 per cent. The early pain is due to extension of the growth to the closely adjoining roots of the lumbar plexus.

The febrile tendency in the earlier stages of the disease, and the hypothermia usual in advanced cases, together with the various other symptoms already enumerated, seem to point directly to a primary excitation by the growth, followed by impairment, of the adrenal functions relating to oxidation and tissue metabolism, according to the conception of these functions introduced by Sajous, Sr.

After emaciation and adynamia have been progressive for some time, a mass can, as a rule, be discovered by palpation posteriorly below the ribs. In infants, however, there may be noticed merely a gradual enlargement of the abdomen with an increasing area of dullness. In long-standing cases, edema (the result of pressure on vessels) may be noted.

On the borderline between benign and malignant growths of the adrenals are the paragangliomas or chromaffin tumors, developing in the medulla of the adrenals or the carotid glands. J. S. Dunn,<sup>63</sup> moreover, has collected from the literature 51 cases of adrenal ganglioneuroma, a malignant tumor developing either in the adrenals or the sympathetic nerve tissues. His own case, that of a boy aged 14, succumbed after a three months' illness featured by wasting, pain in the back, ascites, and marked hepatic enlargement.

*Treatment.* The results of operative work, where removal of the growth is attempted, are apt to be unsatisfactory, the diagnosis being, as a rule, made only at an advanced stage, when metastasis has occurred. If one should succeed in operating sufficiently early to remove the entire disease focus, the opposite adrenal, remaining unaffected, would doubtless prove adequate in maintaining the adrenal functions. Where, in advanced cases, these functions have been so impaired as to give rise to the syndrome of Addison's disease, treatment with adrenal and other products, as described under Hypoadrenia (*q.v.*), might temporarily prove of distinct service.

**Benign Tumors.** Among the recorded types of benign tumors of the adrenals, other than mere hyperplasias, are adenoma, fibroma, lipoma, angioma, and echinococcic cyst. Michon<sup>64</sup> has reported the case of a man of 62 in whom *post-mortem* a large adenoma of the left adrenal was noted. In life, nothing had been noticed save the presence of a tumor in the left hypochondrium and a marked increase in urea excretion in the urine—the latter condition probably an illustration of the important influence of the adrenals on general metabolism. In many instances, benign adrenal tumors exert a peculiar influence on the sex characteristics, particularly inducing in female subjects changes tending toward what Tuffier<sup>65</sup> terms "adrenal virilism." Thus, a woman of 62, in whom a large fibrolipoma of the adrenals was noted at the autopsy, had developed during life—since the age of 30—a thick black beard and moustache, a masculine face and voice, great muscular power, a fondness for hard manual labor, etc., and the clitoris had become so enlarged as to resemble a penile organ. A similar case of hermaphroditism due to an adenoangiolipoma of the left adrenal which had attained the size of a cocoanut is

described by Auvray.<sup>66</sup> Bourcy and Legueu,<sup>67</sup> operating for a large, painful, left-sided abdominal tumor in a woman of 61, found a lymphangiomatous adrenal cyst holding 5 liters of fluid practically surrounding the left kidney. As regards hydatid cysts, Nicaise,<sup>68</sup> among 10,000 cases of this form of parasitism, found *post-mortem* 6 cases of cystic involvement of the adrenals; in none of these had evidences of adrenal impairment been noticed during life.

*Treatment.* This consists of excision for pressure symptoms, and the use of adrenal products and other indicated remedies where adrenal insufficiency results from bilateral involvement.

## DISEASES OF THE THYROID GLAND.

Developing as a bud from the pharynx, which, in turn, grows downward from the posterior portion of the tongue into the neck, the thyroid, in its completed state, consists of a median isthmus and two lateral lobes that the isthmus unites just below the level of the cricoid cartilage. The surgical capsule of the gland, a process of the deep cervical fascia, holds it in close apposition to the trachea, this anatomical feature accounting for the rise of the gland during the act of swallowing and the ease with which the trachea can be seriously compressed and deformed in the presence of thyroid enlargement.

Apart from the so-called pyramidal lobe of the thyroid—an anomalous mass of thyroid tissue which sometimes develops from the thyroglossal duct—accessory lobes may occur anywhere in a triangular area with its base at the margin of the lower jaw and its apex at the root of the aorta, and give rise to retrosternal goiters.

The thyroid tissue is composed essentially of vesicles of different sizes lined with a single layer of cylindrical or cubical cells. These vesicles contain a characteristic colloid material in which the supposed physiologically active thyroid constituent, iodothyronin, is embodied. The gland being ductless, its internal secretory product has been thought to pass into the blood from the lymphatics surrounding it, the vesicles presumably rupturing to discharge the colloid material into the lymph-spaces. Although doubt persists as to the precise route and



rate of absorption or discharge of the colloid, it is nevertheless established that stored colloid may be dissipated from the gland in a relatively short time, and that for months the organ may then remain practically deprived of visible colloid. According to Bensley,<sup>69</sup> a "secretion antecedent" of the thyroid occurs in vacuoles in the outer poles of the cells lining the vesicles, and consists of a solution having properties similar to those of the colloid in the vesicles, but more dilute, and ready in the outer portions of the lining cells to be transported into the vascular channels. The vesicle contents, according to this observer, who studied the structure of the thyroid as modified by diet and drugs, are the result of a second, indirect type of secretion in which the gland products are condensed into droplets having a high content of solids and then extruded into the lumina of the vesicles to be stored for future use when required. Lack of colloid in a given thyroid thus does not necessarily signify a permanent incapacity to secrete, but rather a depletion of the stored material for the time being. Scanty in young children and in parenchymatous goiter, the colloid is unusually abundant in exophthalmic goiter. The amount, it is stated, varies also according to the locality in which the subject resides.

Removal of the thyroid causes pronounced morbid effects, but these have been found to vary according to whether the parathyroids are simultaneously removed or allowed to remain. The difference in the results of thyroidectomy in carnivora and in herbivora was shown by Gley in 1892 to be due to the fact that whereas in the latter two or more of the parathyroids are anatomically separate from the thyroid and hence are not removed in thyroidectomy, in the latter all the parathyroids are so imbedded in the thyroid as to be spared only by dint of special care. Excision of the thyroid gland alone, as Gley showed, does not necessarily cause death. The morbid phenomena it induces are especially severe in young animals, growth being arrested, the bones and epiphyseal cartilages failing to develop, the abdomen becoming protuberant, and enlargement of the sexual glands arrested. Mental development is likewise markedly obtunded, the skin is rough, the hair becomes coarse, shaggy, and lustreless, and the animal dies after a more or less prolonged period of progressive

cachexia. In adult animals, no effects of exclusive thyroidectomy on stature are apparent, growth having already been completed, but marked changes are nevertheless manifest, viz., impaired general nutrition and emaciation, anemia, coarseness of the skin, falling of the hair, hypothermia, etc. All these changes are aggravated, in the female sex, by pregnancy and lactation.

Removal of the parathyroids, alone or in conjunction with thyroidectomy, causes early death, and substitutes for the more gradually developing symptomatology of the latter operation a group of nervous phenomena characterized by a tendency to spasm and convulsions. These disturbances range from tetany to violent tetanic or epileptoid seizures. Death takes place from cramp asphyxia or exhaustion, usually in from three to five days. (See Diseases of the Parathyroids.)

Of interest, further, in comparison with the effects of thyroidectomy are those of an excess of thyroid product. As shown by Ewald, Fenwick, Haskovec, and many others, such an excess induces tachycardia, nervousness, rapid loss of weight, vasodilatation, diuresis, polydipsia, polyphagia, polypnea, glycosuria, hyperthermia, and excessive excretion of nitrogenous wastes, eventually followed by extreme depression, anorexia and vomiting, loss of reflexes, paralyses, convulsions, and death. From the nature of the effects in the earlier stages, coupled with numerous related clinical and experimental observations, the conclusion can hardly be escaped that the thyroid physiologically exerts an important influence on general metabolism and nutrition. That this is actually the case is clearly illustrated by recent accurate determinations of the basal metabolism, which has been shown to be increased in hyperthyroidism to a degree unattained in any other morbid condition, and correspondingly decreased in hypothyroidism.

The precise nature of the active substance present in the thyroid has been the subject of painstaking investigations for many years. Baumann in 1895 announced the discovery of an organic iodine compound, which he termed *iodothylin*, as the chief principle secreted by the gland. Later, however, Oswald, on the basis of careful observations, found reason to deny Baumann's view, and was led to describe the true thyroid principle as an iodized globulin, termed by him *iodthyro-*

globulin. More recently, E. C. Kendall, of the Mayo Clinic, has isolated from the gland a crystalline substance containing no less than 60 per cent. of iodine, and possessing the physiologic activity of the gland itself. Preliminary work directed toward a determination of the structural formula of this substance—termed by Kendall the alpha iodine compound of the thyroid—made it appear<sup>70</sup> to be carbonic acid, in which one of the hydrogen atoms is replaced by diiodo-indol. The absorption of iodine by the thyroid and its elaboration into the thyroid hormone have been subjects of interesting experimental studies by David Marine and his co-workers. Marine<sup>71</sup> observed that living thyroid tissue has an extremely pronounced affinity for iodine, which is rapidly taken up by the gland in whatever form and by whatever method it is administered. The amount of iodine taken up was found to vary, not only according to the size of the gland, but also according to the existing degree of thyroid hyperplasia and the degree of saturation of the thyroid with iodine at the time. With J. M. Rogoff, the same experimenter<sup>72</sup> found that while the storage of iodine in the thyroid from potassium iodide is practically instantaneous, transformation into the specific hormone is much slower, only a small fraction of the iodine taken up having been thus transformed after thirty hours.

That nervous influences are capable of markedly exciting secretory activity on the part of the thyroid seems to have been definitely demonstrated by W. B. Cannon and McKeen Cattell,<sup>73</sup> who studied the activity of the gland through the attending electrical changes by means of the string galvanometer. The sympathetic system proved, in fact, to be a controlling factor in regulating thyroid activity, as Sajous, Sr., urged as far back as 1903. Adrenalin, moreover, was found to cause "quite remarkable action currents," and stimulation of the splanchnic nerve indirectly activated thyroid secretion by causing a discharge of adrenalin from the suprarenal glands.

The potent influence of the thyroid secretion on general metabolism, long ago pointed out by Sajous, Sr.,<sup>74</sup> with ample proof in support, raises a question as to precisely how this metabolic influence, evidently due to Kendall's alpha iodine compound, is exerted. Plummer, quoted by Kendall,<sup>75</sup> from

an extended clinical study of thyroid disturbances in several thousand cases, has been led to conclude that the effects of these disturbances are due, not to perverted function, but to altered rate of normal function; that the stimulating effect of increased thyroid activity is not limited to certain organs or tissues, but is active throughout the body, and furthermore that the stimulating effect is *intracellular*. The senior writer of this article<sup>76</sup> was from the first led to recognize that the combined iodine of the thyroid secretion acts by rendering "the phosphorus of all tissue-cells, and particularly their nuclei, more prone to undergo oxidation by the adrenoxidase of the blood." Chittenden<sup>77</sup> had, indeed, emphasized "the apparent connection between the thyroid gland and phosphoric acid metabolism," pointing out the increased excretion of  $P_2O_5$  upon feeding thyroid extracts to animals and the marked decrease after thyroidectomy. The striking effects of thyroid excess on the nervous system also constitute evidence in this direction, and the rapid loss of fat after thyroid administration in the obese is also thus explainable from the presence in the fat-cells of nuclei rich in phosphorus, the purpose of which is to promote prompt oxidation of the stored fat when the organism requires such additional oxidation.

Sajous, Sr., as previously shown in these pages, has ascribed the influence of the thyroid on oxidation in part to an influence of the adrenals, calling attention to the numerous points of similarity in the symptoms of hypothyroidism and hypoadrenia, and in the secreted products themselves. Of interest in respect to oxidation through the thyroid are the confirmation by Youchtchenko<sup>78</sup> of the presence of catalases and an oxidizing ferment in the thyroid, both of which, moreover, he found also in the red blood-corpuscles; the finding by Albertoni and Tizzoni<sup>79</sup> that thyroidectomy leads to decreased power to fix oxygen on the part of the blood, and the observation of Masoin<sup>80</sup> that the relative amount of oxyhemoglobin in the blood gradually lessens after the same operative procedure. More recently, Burge, Kennedy, and Neill<sup>81</sup> found that thyroid feeding increases the catalase of the blood, while decreasing it in the heart and probably in the fat and skeletal muscles. The increase in blood catalase may account, according to these investigators, for the increased oxidation in thy-

roid-fed animals. Numerous clinicians and experimenters have, indeed, noted that thyroid preparations, as well as pathological hyperthyroidia, are capable of causing through augmented oxidation a rise in temperature of several degrees F. Thiele and Nehring<sup>82</sup> found that thyroid extract increases by over 20 per cent. the oxygen intake, and the carbon dioxide output almost as much. After thyroidectomy, on the other hand, opposite conditions prevail.

From the viewpoint of E. C. Kendall,<sup>83</sup> the alpha iodine compound isolated by him in crystalline form from the thyroid acts in the body specifically by the deamination of amino-acids. The resulting products are then either burned directly into carbon dioxide and water or used for the formation of carbohydrates, fats, etc. The greater the amount of iodine in the cell, the greater the destruction of amino-acids, and ultimately, unless the proteins, themselves constituted of amino-acids, are replenished, they too will be exhausted. The function of the thyroid, according to this view, is "to furnish a catalyzer which regulates the rate of deamination." The colloid matter is looked upon as a vehicle for carrying the iodine catalyzer from the gland to the tissue-cells when a heightening of cell activity is required, and for carrying it later back to the thyroid when the need for it subsides.

Merging into, and scarcely less important than, the property of regulating metabolic changes, is the function of the thyroid in resisting and overcoming intoxications and bacterial infections. Granting oxidation and increase of temperature to be important factors, in a general way, in the destruction of toxic wastes or foreign materials and of bacteria and their harmful products, the thyroid, which when strongly active favors both these conditions, cannot but be considered, *a priori*, a major influence in the protection of the organism.

In 1903, Sajous, Sr.,<sup>84</sup> emphasized for the first time the power of the thyroparathyroid secretion to increase the germicidal and antitoxic properties of the blood. In 1907, Fassin<sup>85</sup> observed experimentally that the administration of thyroid products materially increases the amount of germicidal and hemolytic alexins (complement) in the blood. Clinically, Léopold-Lévi and de Rothschild<sup>86</sup> observed thyroid treatment rapidly to exert a favorable influence in autointoxications



and exogenous infections, including erysipelas. Thyroidectomized animals succumb easily to infections, and their blood serum and urine are abnormally toxic to other animals, suggesting that intermediate wastes or other poisonous materials are not adequately destroyed under these circumstances. Reid Hunt<sup>87</sup> found thyroid-fed mice markedly resistant to poisoning by acetonitrile; Jeandelize and Perrin<sup>88</sup> found thyroidectomized rabbits unusually susceptible to poisoning by sodium arsenate; and Lorand observed a similar deficiency in relation to chloroform narcosis.

Bacteria being relatively rich in phosphorus, as shown in the fact that their ash is largely phosphoric acid, they are all the more vulnerable, from the viewpoint of Sajous, Sr., to the destructive action of thyroiodase—the designation applied by him to the iodine-containing product of the thyroid when combined with the adrenoxidase furnished by the adrenals. From this standpoint, moreover, the secretion of the pancreas is also an effective factor in the autoprotective process; while the pituitary body, connected with the thyroid and adrenals through the sympathetic, from his viewpoint, constitutes a governing center which co-ordinates their secretory activities. Palmer,<sup>89</sup> extirpating the main thyroid body in pigs, observed that a degree of hypothyroidism insufficient to cause marked changes in the physical appearance of the animal yet sufficed to lower considerably the resistance to infection, as well as to impair the functions of reproduction.

### THYROID INSUFFICIENCY (HYPOTHYROIDIA).

While the manifestations of thyroid insufficiency are observed in their most intense form in true myxedema, the latter in its fully developed condition is relatively rare. Far more frequent are the cases of incomplete or "fruste" myxedema, which complicates pathogenically many of the diseases met in daily practice, and to which the term hypothyroidia (hypothyroidism; chronic benign hypothyroidia—Hertoghe) may conveniently be applied.

An essential fact to be borne in mind in this connection is that the symptomatology of hypothyroidia does not, as a rule, embody the cardinal myxedemic symptoms. The mani-

festations of hypothyroidia oftenest encountered, singly or in combination, comprise severe occipital and interscapular pain, obesity with supraclavicular pads of fat, low body temperature, loss of hair and teeth, lassitude, inveterate constipation, and mental torpor. In children, these manifestations may be sup-

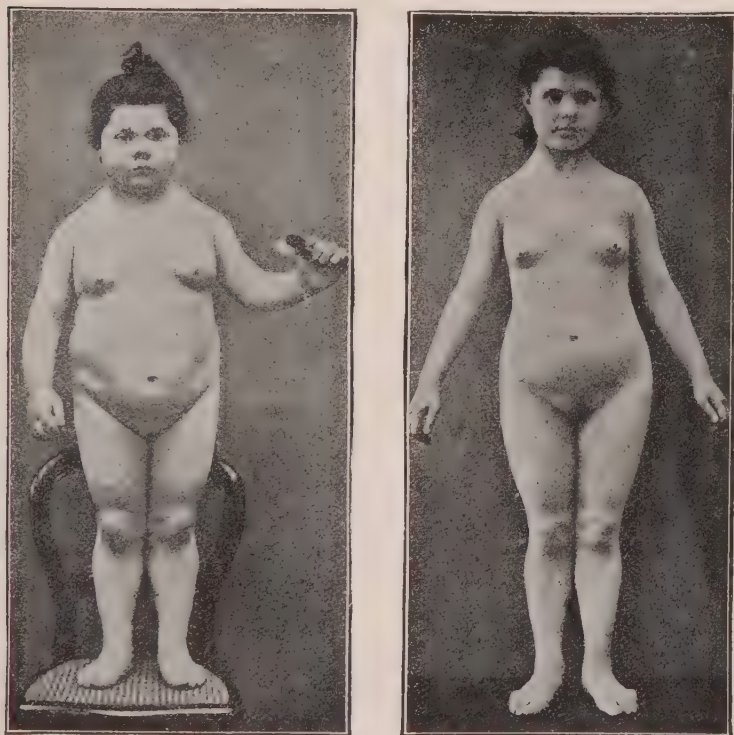


Fig. 1.—Hypothyroidia. Physical development under thyroid treatment. (*Léopold-Lévi and de Rothschild.*)

plemented by slow physical, mental, and irregular skeletal development, enlargement of the lymphatic glands, and occasionally enuresis.

Headache or backache frequently constitutes the chief complaint. Somnolence on rising, with a subjective feeling of improvement as the day wears on; chilliness, due to deficient metabolic activity, and a fondness for stimulants, are also suggestive. Other features include premature ageing and gray-

ness of the hair; a tendency to occipital alopecia, characteristically in the lateral portions of the eyebrows; dryness of the hair, a waxy hue of the facial skin, and puffy eyelids. Dyspnea or oppression on exertion, palpitations, a tendency to cardiac dilatation, anemia, early loosening and caries of the teeth, bleeding and receding gums, persistent congestion of the mucosæ of the upper respiratory tract, passive enlargement of the liver, and a tendency to varicose and calculous disturbances, oliguria, and flat-foot are not infrequent manifestations.

In the female sex, amenorrhea, metrorrhagia, or dysmenorrhea may be noted, and the uterus is often found in retroflexion. Pregnancy and lactation, exciting the thyroid to increased activity, may markedly improve the condition for the time being, or in occasional cases permanently. On the other hand, lactation may at times, by imposing exhausting activity on the adrenals, lead temporarily to increased pallor, a tendency to edema, and intellectual torpor. In males, impotence, spermatorrhea, and prostatic hypertrophy may be observed.

Imperfect circulation in the special sense organs may lead to hallucinations of sight or various forms of tinnitus. The mind, even in the milder cases, is apt to be slightly obtuse, and a melancholic tendency is frequent. Occasionally there is maniacal excitement, probably due to inadequate destruction of wastes in the blood.

The origin of hypothyroidia, which may occur at any period of life, is sometimes hereditary; the chief causes in such cases being syphilis, alcoholism, and the gouty diathesis. Pregnancy may in this form lead to permanent improvement by stimulating the functional activity of the organ and causing it to hypertrophy. The acquired form of hypothyroidia not infrequently originates in some acute infectious disease, causing interstitial and parenchymatous changes in the thyroid which lead later to sclerosis and atrophy. Excessive repetition of pregnancy and thyroid traumatism are also at times causes of acquired hypothyroidia. In the aged the condition is to some extent physiological, all the ductless glands tending toward retrogression in this period—a condition probably in itself essential in the causation of senility.

## TREATMENT.

The chief remedy is manifestly dried thyroid (*Thyroidesum siccum*, U. S. P.), relief being contingent, however, upon suitable dosage. The residual secreting power of the gland varying in different cases, care in the adjustment of the dose is a necessity. In adults, 0.06 gram (1 gr.) of the dried gland three times a day during meals is usually a sufficient initial amount. Later this may be increased gradually to 0.12 gram (2 gr.) three times daily. More than this is seldom required, and in some cases but 0.03 gram ( $\frac{1}{2}$  gr.), or even less, three times a day is sufficient. Excessive dosage in any given case is likely to bring on headache, pain over the kidneys and in the joints, muscles, and liver, anorexia, a rapid pulse, a rise in temperature, a tendency toward fainting, tremor, and an increase of any pre-existing dyspnea. Where there is marked anemia, dried suprarenals 0.12 gram (2 gr.), and Bland's pill-mass, 0.06 gram (1 gr.), may be advantageously combined with each dose of thyroid substance. Properly to relieve constipation, high injections of saline solution two or three times a week may be required at first, after which glycerin suppositories may be used. For purgation by mouth, salines should be given preference. As a rule, the thyroid medication will have to be continued indefinitely, possibly in reduced dosage.

Hemorrhage from the uterus in 6 cases, including 3 of excessive menstruation, the condition being ascribed in all to thyroid insufficiency, was successfully treated by Salzman<sup>90</sup> by dried thyroids in the dosage of 1 to 3 5-grain (0.3 Gm.) tablets a day.

Among men in active military service, Blanc<sup>91</sup> has observed very many examples of thyroid disturbance, including some of insufficiency of this gland. As Petzetakis<sup>92</sup> had previously pointed out, the oculocardiac reflex is enormously intensified in hypothyroidia, the sympathetic being left without its normal stimulation from the thyroid, ascendancy of the autonomic nervous structures and vagotonia resulting. Blanc noticed that under either thyroid or parathyroid medication, or both, the balance was restored and the abnormal oculocardiac reflex disappeared.

**MYXEDEMA (PROGRESSIVE HYPOTHYROIDIA).**

This condition arises as a result of marked or complete hypothyroidia coming on at any period of life after puberty, and, as an expression of the maximum loss of thyroid function, is characterized by general deficiency of oxidation and catabolism, causing hypothermia, infiltration and swelling of the cutaneous tissues, an increase in body weight, dryness of the skin, pronounced asthenia, and mental torpor.

Among such patients the almost continuous suffering from cold, except in hot weather, leads to the use of an abnormal amount of covering. The temperature is always below normal, and, the circulation as well as the processes of oxygenation being impaired, the least exposure to cold induces cyanosis of the peripheral tissues. The infiltrated cutaneous tissues are elastic and firm, but do not pit on pressure, as in true edema. The abdomen becomes pendulous, the hands thickened, and the nails brittle and thin, sometimes ridged or atrophied.

The skin in myxedema often becomes rough and scaly. Patches of pigmentation, varying from yellowish-brown to actual bronzing, and suggesting participation of the adrenals in the functional torpor, are sometimes observed. The hair loses its luster, becomes coarse and brittle, and finally falls out. The facial expression is mask-like, owing to the cutaneous infiltration. All the mucous membranes similarly become pale and tumefied; the condition of the teeth and gums goes from bad to worse; and the attending stomatitis and salivation result in dribbling from the corners of the mouth. Enunciation is imperfect, and the voice coarse, nasal, and low-pitched. Constipation alternates with diarrhea, and there is a profound distaste for meat.

The mental state is characterized by somnolence, apathy, amnesia, and irritability. Exhaustion upon slight exertion is the rule. Sensation is impaired, and the finer movements imperfectly performed. Headache and pains in the muscles and joints are common. The special senses are obtunded or perverted; and hemorrhages, apparently due to poverty of the blood in fibrin ferment, are common. Urea excretion is diminished.





Fig. 1.



Fig. 2.

THYROID TREATMENT IN MYXEDEMA. [*Hertoghe.*]

Fig. 1. True myxedema.

Fig. 2. The same patient after thyroid treatment.

[*Bulletin de l'Académie Royale de Médecine de Belgique.*]



In contrast with simple hypothyroidia, myxedema is, in a sense, progressive, death eventually taking place from exhaustion or intercurrent infection. As a rule the condition lasts six to twenty years; but occasionally, especially in young adults, it may run its course in six months. An acute type of myxedema, with death within a few days, has also been met with.

Myxedema occurs about six times as frequently in women as in men. The main causes are rapidly repeated child-bearing, the menopause, worry, mental shocks, and injuries, especially to the head. Where a familial influence is operative, tuberculosis, neuroses, syphilis or alcoholism is apt to be found in the patient's antecedents.

A distinct reduction in the size of the thyroid gland, characteristically the seat of atrophic change, occurs in the vast majority of cases of myxedema. At times, however, the gland is actually enlarged at first, then gradually atrophies irregularly.

#### TREATMENT.

Remarkable results are obtainable in this condition by thyroid feeding. Six centigrams (1 gr.) of dried thyroid three times a day is sufficient at first, but this may later be gradually increased to 2 grains. Restoration of the body temperature to normal is a useful guide as to the amount actually required. If the temperature rises above normal, the dose should be reduced. The pulse-rate should also be watched, an increase of 15 beats per minute indicating a reduced dose. Exertion is to be avoided during the treatment, especially at first, lest syncope suddenly supervene. To maintain the improvement, the remedy obviously must be continued throughout life, unless thyroid grafting is performed and turns out successfully. (See Infantile Myxedema, Treatment.) Where the heart is considerably dilated, small doses of digitalis or of dried pituitary or suprarenal will materially hasten recovery.

#### INFANTILE MYXEDEMA (CRETINISM).

Thyroid insufficiency occurs, in this condition, before the body growth is complete; there are added, therefore, to the morbid phenomena of myxedema in adults other signs pertaining to the developmental period. The disease is thus char-

acterized by stunted growth, the cretinic facies, with flattened nose and thickened lips and tongue, a harsh skin, and more or less pronounced idiocy. The abdomen projects forward considerably, not infrequently exhibiting an umbilical hernia. The legs are short and bowed, and the hands broad, with stiff and pudgy fingers. As in the myxedema of adults, various manifestations of subnormal oxidation occur. All the muscles being relatively atonic, the child is feeble, wobbles in walking, and may even be unable to stand or hold up its head. Constipation is the rule, interrupted by occasional attacks of diarrhea. The genitals are usually imperfectly developed, though occasionally, on the contrary, the sexual instincts are enhanced. Menstruation often fails to appear, or may be menorrhagic.

From the mental standpoint, the cases have been divided by the brothers Wenzel into three groups: the cretins, unable to speak; the semicretins, simple-minded, but able to speak in an imperfect manner; and the cretinoids, possessing some intelligence, but presenting physical evidences of cretinism.

The cases are also necessarily divided into the endemic and sporadic types, the former constituting frequently a family disease, observed in groups of cases in special localities, and ascribed to some chemical substance or micro-organism peculiar to the waters used in those districts. In a certain proportion of cases, it is believed, endemic cretinism may be congenital. In a considerable number of the endemic cases, a more or less voluminous goiter exists.

Sporadic cretinism is met with in localities free of endemic, grouped cases, and among healthy families. The condition is ascribed to some thyroid lesion caused by an acute febrile disease or intoxication, either before or after birth. Among the most frequent infectious causes are typhoid fever, scarlatina, pneumonia, and pertussis.

#### TREATMENT.

Growth arrested by cretinism is restored with surprising rapidity by thyroid treatment, the child developing sometimes by over an inch per month, until the stature normal at the corresponding age has been attained. As regards mental development the results, while remarkable, may not be com-



Fig. 1.



Fig. 2.

THYROID EXTRACT IN CRETINISM. [J. B. McGee.]

Fig. 1. Cretinic idiot 7 years old when thyroid treatment was begun.  
Had ceased to develop when 3 years old.

Fig. 2. Changes after one year's treatment. Growth,  $6\frac{1}{2}$  inches.

[Cleveland Medical Gazette.]





plete unless the treatment has been started early. Where an interval of years has elapsed between the onset of cretinism and the beginning of treatment, the intelligence regained does not, as a rule, attain that of the normal child.

As in adult myxedema, careful adjustment of dosage is necessary. Excessive dosage not only entails danger of sudden heart-failure, but may also lead to softening of the bones, owing to an unduly rapid growth—a tendency to be guarded against, if the bones begin to yield, by the application of suitable braces. At times toxic symptoms may develop in a sudden, unexpected manner, the drug being in a sense cumulative in its action. Seemingly, such results are more likely to be produced when the glandular preparation used is old. An infant can, as a rule, be given 0.03 gram ( $\frac{1}{2}$  gr.) of dried thyroid once daily, a child of two years, twice daily, and older children, 3 times a day, or 0.06 gram (1 gr.) twice daily. As recommended by Morton, the child should, as a precautionary measure, be kept recumbent after the last dose, given late in the day. When the normal body temperature has been reached under the influence of the treatment, 0.06 gram (1 gr.) of dried thyroids on retiring will, as a rule, be sufficient to prevent recurrence. Danger signals necessitating a reduction in dosage comprise a rapid pulse, dizziness, pains in the back and extremities, general weakness and a syncopal tendency, or, ultimately, nausea and vomiting, a pronounced rise of temperature, and collapse. In cretins who have reached adult age before treatment is started, improvement is but slight, or may be *nil*. Again, in sporadic cases the improvement is more marked than in the endemic, irreparable injury having often been produced in the latter type before treatment, while in the former the earliest, most important stages of growth may have been passed before the onset of the disease.

To antagonize any accompanying tendency to softening of the bones, the administration of syrup of lactophosphate of lime in teaspoonful doses may prove a useful adjuvant to thyroid treatment. Dried thymus gland 0.3 gram (5 gr.) thrice daily, is also of advantage.

To obviate the necessity of thyroid medication throughout life, thyroid grafting has been rather widely experimented with, though, on the whole, with disappointing results. Ac-

cording to Cristiani,<sup>93</sup> the grafts must be of normal human thyroid tissue, and be introduced only in very vascular subcutaneous tissue, in small but multiple masses. Charrin and Cristiani<sup>94</sup> obtained, however, good results also with sheep's thyroid. In a series of cases the latter observer<sup>95</sup> noted distinct improvement in 60 per cent., remarkable results in 34 per cent., and no improvement in 6 per cent. Kummer<sup>96</sup> has reported a successful autograft of normal segments of the thyroid under the skin over the right acromion in a woman with a large goiter, in whom both lobes of the goiter had been almost completely removed. Charles Goodman<sup>97</sup> concluded from experimental work in dogs that as yet no means were available for prolonging indefinitely the life of an entire organ transplanted from one animal to another. He agrees with Carrel, Lexer, and others, however, that autotransplantation is practicable. In his experiments the arterial supply of the transplant was provided for by suture of an attached segment of carotid into the carotid of the host, and the venous supply by end-to-end suture of the thyroid vein with the central end of the external jugular of the opposite side. In autotransplantation, the author thus succeeded in two consecutive instances in retaining the thyroid gland in its normal state microscopically. Among the homotransplantations there were a few instances in which parathyroid tissues remained normal while the thyroid showed evidences of hemolysis. Kocher,<sup>98</sup> while recognizing that thyroid transplantation acted even more effectually and promptly than thyroid medication in myxedematous states, concluded that a single transplantation was insufficient, and asserted that a possible method of obtaining a permanently active transplant would be to decrease the immunity of the recipient, such immunity tending to hasten destruction of the transplanted tissue.

### MYXEDEMATOUS INFANTILISM.

In this condition the deficiency of thyroid function is manifested particularly in a persistence of the physical and mental characteristics of childhood rather than in the actual idiocy and dwarfism of the cretin. While the most severe cases are virtually instances of mild cretinism, and do exhibit

some of the physical characteristics of this disorder, an essential feature of the average case is the tendency toward retention, both as to ideas, judgment, and emotions, of the intellect of much younger subjects. Physically, the heart is excitable, varicose disorders and hemorrhagic tendencies are frequent, and the genitals may remain of rudimentary size. In the mildest forms the signs of myxedema are hardly discernible, and physical development may even surpass the average. In males, however, the general conformation frequently resembles that of the female.

#### TREATMENT.

Thyroid products constitute the chief therapeutic resource, as in cretinism. The younger the patient, the more likely is improvement to occur. After puberty, the results, from the standpoint of the mentality, are seldom satisfactory.

#### THYROIDITIS.

Acute hyperemia or actual inflammation of the thyroid gland is generally a result of some infectious disease. In the presence of mere hyperemia, slight swelling of the gland, with some tenderness and dysphagia, are alone noticed. In acute thyroiditis, however, severe local as well as general manifestations occur, a chill marking the usually sudden onset, which is followed by considerable swelling of the gland, marked dysphagia and radiating pains, dyspnea from tracheal compression, and possibly paralysis of the recurrent laryngeal nerve or edema of the glottis. Fever may be high in spite of the absence of suppuration, and the tachycardia typical of thyroid overactivity has also been observed, independently of fever. In a few days, as a rule, the morbid condition subsides; resolution without suppuration occurs in 40 per cent. of the cases. Where suppuration does develop, the abscesses are, as a rule, multiple, each, however, tending to break through the adjoining soft tissues, thus leading to confluence, rupture through the skin, or purulent infiltration of surrounding structures, at times leading to dangerous complications, such as pneumonia and pyemia. The abscesses bleed easily, severe capillary hemorrhages sometimes constitute a complication.

The infections leading to acute thyroiditis comprise such disorders as diphtheria, typhoid fever, scarlatina, mumps, tonsillitis, erysipelas, pneumonia, measles, pertussis, rheumatic fever, puerperal fever, etc. A toxic thyroiditis due, *e.g.*, to iodids, is also recognized. The wealth of the thyroid tissue in vascular channels is such that even in the absence of actual thyroiditis, a high blood-pressure, such as that of fevers, may lead to interstitial hemorrhages. Either through subsequent interstitial sclerosis or because of degeneration of the epithelium in thyroiditis, the thyroid functions may become so impaired as to lead to marked evidences of hypothyroidia, or even typical cretinism. On the other hand, as pointed out by Theisen,<sup>99</sup> after thyroiditis complicating acute tonsillitis, hyperthyroidism may develop.

Chronic thyroiditis may follow the acute type, resolution having been incomplete. Oftener, however, it accompanies such chronic disturbances as syphilis, tuberculosis, actinomycosis, and echinococcus disease. The resulting ultimate reduction in thyroid activity is a most common cause of hypothyroidia.

A parasitic form of thyroiditis, due to a flagellate organism (*Schizotrypanum cruzi*), is frequently encountered in the State of Minas Geraes, Brazil.

#### TREATMENT.

In infectious diseases the thyroid should be carefully watched, and where local pain, tenderness, or swelling develops, cold compresses should be applied, not only to cause contraction of the vessels beneath, but to reduce the local temperature, and thereby also the activity of the protective principles concentrated in the thyroid, which, in thyroiditis, probably exceed the limits of beneficial action, and lead to autolysis of the gland tissue. Saline solution, by mouth, rectum, or the subcutaneous route, is also of importance in these cases. A high blood-pressure, tending to perpetuate thyroid congestion, may at times be advantageously lowered with such agents as chloral hydrate and veratrum viride. Rest is, as in other local inflammations, an important measure, and leeching may exert a useful decongestive effect. For dyspnea of alarming degree, tracheotomy may be required,



Suppuration occurring in approximately a half of all cases, the frequent advisability of surgical intervention must be borne in mind. The presence of pus being difficult of demonstration, exploratory puncture may prove of assistance. According to Kocher,<sup>100</sup> if incision does not result in rapid recovery, multiple abscesses should be suspected; persistence of a sinus points to extensive necrosis, and the affected half of the gland should then be excised.

In chronic thyroiditis leading to hypothyroidia thyroid treatment should be instituted. Removal of chronically diseased thyroid tissue may be advisable, particularly where dyspnea is troublesome. De Massary<sup>101</sup> has reported a case of chronic syphilitic thyroiditis, with marked impairment of the intellectual powers, in which thyroid medication restored the mental condition, and mercury (begun only six months later) reduced the thyroid gland, previously large and soft, to more nearly normal dimensions.

### THYROID OVERACTIVITY (HYPERTHYROIDIA) AND EXOPHTHALMIC GOITER.

Some of the physiological disturbances awakened by excessive thyroid functioning, in particular the marked heightening of general metabolism and oxidation, were referred to at the beginning of this section. By the term hyperthyroidia—or larval, “fruste” exophthalmic goiter—may conveniently be designated those instances of thyroid overactivity in which certain cardinal symptoms of true exophthalmic goiter, viz., exophthalmus and goiter, are lacking. In its etiology, pathology, symptomatology, and medicinal treatment, however, hyperthyroidia is very similar to exophthalmic goiter—Graves’s, Basedow’s, or Parry’s disease.

That exophthalmic goiter is due to excessive activity of the thyroid can no longer be doubted. The clinical and therapeutic aspects of the condition render it advantageous to recognize three stages: (1) the *sthenic* or *erethic* stage, in which excessive oxidation and abnormally active cellular metabolism prevail; (2) a *transitional* stage, in which a gradual restriction of the thyroid function, due to sclerotic changes and atrophy from overwork, is initiated; and (3) the

*asthenic* or *myxedematous* stage, in which the increasing lack of thyroid function is definitely manifest in symptoms indicative of hypothyroidia, and in which death occurs.

In the *sthenic* stage the excessive metabolism, which, according to views set forth by the writer 15 years ago,<sup>102</sup> is in part due to overactivity of the adrenals in concomitance with that of the thyroid, seems to involve with especial sever-



Fig. 2.—Graves's disease with pronounced thyroid enlargement and exophthalmus. (From Da Costa's Physical Diagnosis. Copyright, W. B. Saunders Co.)

ity the structures rich in phosphorus, including the nervous system, Chittenden<sup>103</sup> having laid stress on the marked increase in phosphoric acid excretion in this condition. Manifestations of such excessive phosphoric metabolism are: the characteristic nervousness and tremor of exophthalmic goiter, and the convulsive movements sometimes noticed. That oxygen consumption and carbon dioxid elimination are often augmented to a surprising degree has been definitely established, and, concomitantly, there is frequently an increase of the body temperature to 100° or 101°, or a recurrent actual febrile

state, in which even such temperatures as  $107^{\circ}$  to  $110^{\circ}$  have exceptionally been recorded. According to DuBois,<sup>104</sup> who studied metabolism in 11 patients with a respiration calorimeter, heat production gives the best indication of the severity of the disease in a given subject. Some of his cases showed an increase of 75 per cent. or more above normal in heat production; the moderately severe and most of the mild cases, an increase up to 50 per cent.



Fig. 3.—Graves's disease without exophthalmus. (From Da Costa's Physical Diagnosis. Copyright, W. B. Saunders Co.)

Other well-known manifestations of the erethism of this stage of the disease are the tachycardia, the abnormal irritability of the alimentary canal (manifested, *e.g.*, in gastric hyperesthesia and diarrhea), and the general vasodilation and low blood-pressure, apparently due, at least in part, to the special excitation of the depressor nerve noticed experimentally as an effect of thyroid products by Cyon many years ago. Seemingly dependent on this general tendency to vascular atonicity are the flushing of the skin, epistaxis, hemorrhagic areas in the mucous membranes, edemas in the eyelids and lower limbs, exophthalmus, etc.

While some cases of exophthalmic goiter are spontaneously recovered from, and others soon succumb in the erethic stage after suddenly entering a rapid downward course, very many come eventually into a phase in which there seems to be considerable improvement. The skin moisture, the sensations of heat, and the nervous phenomena, including tachycardia, the emaciation, etc., tend to abate. The goiter may recede somewhat, and exhibit palpable nodules, indicative of localized sclerotic changes in the thyroid parenchyma. From this (the transitional stage) the case then passes into the third phase, that of myxedema, with the customary manifestations of which are coupled certain indications of exhaustion of the adrenals—in particular, pigmentations of the skin. To the ordinary signs of myxedema are added greater cardiac weakness and dilatation, owing probably to exhaustion of and permanent injury to the myocardium during the sthenic stage. To some intercurrent disease, or a cachexia terminating in heart failure, the case ultimately succumbs.

Participation of the thymus in the pathogenesis of exophthalmic goiter has been emphasized of late. In about 82 per cent. of cases examined *post-mortem*, in whom death had been due to intercurrent disease, a persistent thymus has been found, while, according to the observations of Haberer, injection of thymus gland extract is capable of inducing thyrotoxis. Bircher has produced typical exophthalmic goiter symptoms by implanting fresh hyperplastic thymus tissue intraperitoneally. Where the goiter is small, dyspnea on exertion, with a sensation of pressure behind the manubrium, or dysphagia, with the "feeling" of a lump in the same situation, are indications of thymic involvement—a condition confirmed by x-ray examination and the finding of dullness over the thymic area. An added danger where the thymus is involved is that sudden death may occur during or even some days after an operation on the thyroid.

In the etiology of exophthalmic goiter, two factors stand out above all others, viz., the toxic or infectious and the nervous. The former group includes not only bacterial toxins, but also autogenous toxic substances absorbed from the alimentary canal because of imperfect digestion of nitrogenous foods. Ether anesthesia, and violent, prolonged physical work

have also occasionally been causative. In the nervous group belong anger, fright, and other emotions, traumatic shock, blows upon the head, etc. Such causes may be held operative through disturbance of the sympathetic, which governs the caliber of the arterioles of the thyroid. As Cannon<sup>105</sup> has shown, the thyroid, like the adrenals, has an emergency function, serving at critical times to accelerate metabolism to an unusual rate. Like the adrenals, furthermore, the thyroid was found by Cannon to receive impulses through the sympathetic system. The pituitary body embodying, according to the senior writer, a governing center of the sympathetic system, the possibility that this organ might be the primary seat of the nervous disturbance in these cases has been suggested. Apparently in many instances a toxic and an emotional or traumatic factor co-operate in the etiology of exophthalmic goiter. The European war has afforded numerous examples of the disease arising either through trauma to the head, physical or mental overwork, intoxication from poor food or water, and infections such as dysentery and typhoid or paratyphoid fever. Pietrowicz<sup>106</sup> rightly laid stress on infectious conditions in the mouth, teeth, tongue, nose, tonsils, pharynx, and larynx as a factor in the disease.

#### TREATMENT.

An important feature of the medical treatment is to promote contraction of dilated vessels, especially those of the thyroid gland itself, and those of the postorbital region. Probably to such an action are to be attributed the good results obtained by Huchard, Paulesco, and others from the administration of ergot in combination with quinin. An effectual mode of applying this treatment is that recommended by Forchheimer, who gave in a capsule 0.06 gram (1 gr.) of ergotin (watery extract of ergot) and 0.3 gram (5 gr.) of neutral hydrobromid of quinin after each meal. Where no signs of cinchonism develop, a fourth capsule may be taken at bedtime. To obviate unpleasant effects, as well as to reduce the central erethism, 1.3 gram (20 gr.) of sodium bromid at bedtime may also be given with advantage, together with 0.6 gram (10 gr.) of chloral hydrate where sleeplessness is further complained of. In some highly nervous women whom quinin disturbs, acet-



phenetidin in 0.3-gram (5-gr.) doses, gradually increased to 0.6 gram (10 gr.) three times a day, may be used with benefit to supplement the bromid and chloral.

Probably acting in a similar manner is pituitary extract which, according to Hallion and Carrion,<sup>107</sup> exerts an intense vasoconstrictor action on the thyroid. Pal<sup>108</sup> has reported a case improved by pituitary injections, while Richter<sup>109</sup> has had good results in several cases from daily administration of 3 to 5 5-grain (0.3 Gm.) tablets of extract of anterior lobe of the pituitary.

According to Kocher and others, sodium phosphate is of considerable value in exophthalmic goiter. Calcium salts, sodium sulphate or glycerophosphate, and lecithin in an alcoholic solution have also been recommended.

Rest is a measure of the utmost importance in all but very mild cases. A. J. Ochsner's rules<sup>110</sup> as to rest and diet in these cases after operation are likewise applicable with advantage in the purely medical treatment of the disease. All excitement or irritation is to be avoided, and the patient should get plenty of rest, going to bed early and taking a nap after lunch. Nothing irritating to the nervous system should be eaten or drunk. Tobacco should be avoided, and very little meat used. Meat broths should be eschewed. Milk and foods prepared with milk are to be freely taken; likewise cooked fruits and vegetables, or very ripe, raw fruits. Eggs, bread, butter, toast, rice, and cereals are all permitted. Good drinking water should be used freely, if good water is not available, the water to be drunk should be boiled for twenty minutes, or distilled water used.

An essential measure is a careful search for any underlying infectious or toxic factor, and if such be found, its eradication. Tonsillectomy is often indicated. Where there is a clear history of acute rheumatism, sodium salicylate in 0.6-gram (10-gr.) doses three times a day will often satisfactorily counteract the hyperthyroidia. Pyorrhea alveolaris appears to be a very frequent cause. For many toxic underlying states, saline enteroclysis at 108° F., after a cleansing enema, is an eliminatory measure of considerable value.

In cases due to pregnancy, the menopause, or ovarian underdevelopment, in which the disorder seems to represent an

ineffectual attempt of the organ to neutralize accumulated wastes in the blood, dried thyroids, 0.06 gram (1 gr.) thrice daily, seem not infrequently to be productive of good—though contraindicated elsewhere—by compensating for the insufficient functional capacity of the thyroid. Thymus gland was accidentally found by Owen to be serviceable in exophthalmic goiter, provided the case is not one with concomitant thymic enlargement. The dosage is 1 to 3 0.3-gram (5-gr.) tablets three times a day during meals. S. Solis-Cohen<sup>111</sup> has recommended thymus in conjunction with pituitrin, the latter in intramuscular injections.

Such preparations as Möbius's antithyroidin, Ballet and Enriquez's dog serum, Rogers and Beebe's serum, and thyroidectin have not yielded convincing results in our hands.

Where measures such as those already described fail, much may be done by the injection of boiling water into the thyroid, as suggested by M. F. Porter.<sup>112</sup> The large, graduated glass syringe used in this procedure is boiled in the water used for the injection. The skin, after being cleansed, is anesthetized by Schleich's method. By the use of a long needle both the right and left lobes and the isthmus may be injected through 1 skin puncture, made in the median line. The amount of water injected by Porter ranged from 2.5 to 15 mils (40 to 230 *m.*). The measure causes an immediate destruction of thyroid tissue and colloid, and, after a short period of irritative reaction, benefit is rapidly noticed. At least one death having followed the procedure—albeit in a most desperate case—other similarly acting but safer methods have been sought, and one has apparently been found in the use of injections of a sterilized 30 to 50 per cent. solution of quinin and urea hydrochlorid, which, while seemingly devoid of danger, causes effectual necrosis of thyroid tissue and subsequent fibrosis, thus cutting down as much as may be desired the secreting tissue. Series of bi-weekly injections of such a solution will often produce what seems to be a permanent cure, even in very pronounced cases. L. F. Watson<sup>113</sup> has, among others, had good results from this method of treatment, and our own correspond with his.

Röntgen ray treatment of exophthalmic goiter may now be availed of without risk of burns since the introduction of pre-

cise methods of measuring the dose of rays administered. According to C. A. Simpson,<sup>114</sup>  $\alpha$ -ray treatment is always the procedure of choice where thymus enlargement is suspected, as it will quickly and painlessly atrophy the thymus gland. Fischer,<sup>115</sup> treating 94 cases with the rays, obtained positive benefit in 77 to 80 per cent., no improvement occurring in the remainder. In 15 cases all objective and subjective signs and symptoms of the disease subsided completely under the treatment. Pfahler and Zulick<sup>116</sup> believe that by routine trial of  $\alpha$ -ray treatment many thyroid operations can be avoided, but warn that the treatment must not be too prolonged, or hypothyroidia may be induced. Increased weight and a decrease in the pulse-rate are the first signs of improvement, and nearly always occur. The goiter and exophthalmos are the last manifestations to improve, and in many cases they remain uninfluenced.

In the transitional stage of exophthalmic goiter signs of myxedema are apt to be present, and treatment for their correction may have to be instituted. In the myxedematous stage the necessity for thyroid treatment will be obvious, together with digitalis or strophanthus to antagonize the marked tendency to cardiac failure.

*Surgical Treatment.* The reports of careful clinicians seem to indicate that a cure of the condition can be encompassed by non-operative methods in from 80 to 90 per cent. of cases. From the standpoint of the average surgeon, on the other hand, exophthalmic goiter is largely a surgical disease, in which prolonged attempts at curative medical treatment constitute a mere waste of time. Doubtless, an intermediate ground is more in keeping with the true necessities of the condition than either extreme, but an important fact to be realized is that most, if not all, cases are secondary to some underlying toxic, infectious, or nervous condition, removal of which, whether by medical or simple surgical means (tonsillectomy, for example), will, if accomplished sufficiently early, remove all need for the more serious interventions on the thyroid gland itself.

The risk, slight but not negligible, attending operations on the thyroid is illustrated in the mortality percentages mentioned by Berkman,<sup>117</sup> referring to the experience at the Mayo

Clinic from 1910 to 1915. In these successive years, respectively, the operative mortality was 4.8, 3.18, 2.6, 3, 2.89, and 2.63 per cent., the cases in question being all instances of unmistakable hyperthyroidism. As regards the ultimate results from operative work, Judd and Pemberton<sup>118</sup> have published a statistical study of cases operated upon at the same clinic in 1909, and traced subsequently. Of 121 cases, 55, or 45 per cent., were cured, while 22, or 18.1 per cent. still had some traces of the disease. In 5 of the cases there was but slight improvement, and in 8, no benefit. The average period required to effect a cure was no less than 17.9 months.

On the whole, while admitting that in a certain percentage of cases, late in coming under treatment, indications may exist for surgical treatment as soon as suitable preparatory measures can be completed, our own conviction has steadily been increasing that all but a very small proportion, probably 5 per cent., are curable without surgical measures. The field for the latter, under these conditions, is largely limited to cases in which, after prolonged, careful modern medical treatment, it is evident that the amount of secretion cannot be sufficiently cut down in any way other than actual removal of the gland. Even this contingency seems somewhat remote, presupposing that all non-operative measures be unrelentingly applied.

With such destructive procedures as boiling water and quinin and urea injections available, not to speak of the *x*-rays, there appears little reason why, if such measures be repeated until widespread destruction of gland-tissue has been produced, the excessive thyroid function causing the disease should not be mastered without surgical intervention.

At the Mayo Clinic it has been customary, in dealing with these cases, first to ligate the left superior thyroid vessels under local anesthesia. If no reaction follows, it is considered safe, after a week, to remove the right lobe of the gland, or more, if indicated. In the event, however, of a considerable reaction, marked by exaggeration of the tachycardia, vomiting, diarrhea, and restlessness, ligation of the right superior thyroid vessels is also performed, after subsidence of the reaction. After such a second ligation, it is considered advisable to wait about three months before thyroidectomy, the case being usually changed in this time from a questionable or

serious surgical risk to a good one. In some cases, furthermore, a complete cure may follow the two ligations, rendering the thyroidectomy unnecessary. Where a partial thyroidectomy fails to cure, resection of the remaining part of the gland, it is asserted, will often be productive of much good, the cure being not infrequently rendered complete thereby. The risk of provoking myxedematous symptoms through excessive removal of thyroid tissue is, of course, always to be borne in mind. Sometimes, on the other hand, symptoms of hyperthyroidism return after a period of health, through transformation of residual tissue into a goiter.

Preliminary preparation for an operation on the thyroid, even an arterial ligation in the serious cases, is an important factor in helping the patient safely to withstand the surgical procedure. Such preparation includes, in particular, absolute rest, heart tonics, diuretics, and sometimes the *x*-rays. The latter have proven serviceable in helping patients through acute attacks, previous to operation. Where such acute symptoms are present, thyroidectomy must be postponed until they pass off. Irregularity and varying tension of the pulse, diarrhea, edema of the hands and feet, sleeplessness, and paroxysms of gastric pain are all conditions rendering postponement of operation advisable.

Removal of the thymus at operation has been advised by some, owing to the frequent causal relationship of this organ to the disease. In Haberer's experience, a combined partial operation on both thyroid and thymus, in the cases with persistence of the latter organ, has given results superior to those attending thyroidectomy alone. In the *x*-rays, however, we have a procedure which will effectually reduce the thymus in these cases before the thyroidectomy is undertaken, thus removing the occasion for dealing surgically with the thymus, and also lessening the risk from the thyroid operation.

### GOITER.

This protean condition, the essential expression of which is an enlargement of the thyroid gland, and which has also been termed struma or bronchocele, differs from exophthalmic goiter, in general, in the absence of systemic evidences of thy-



roid intoxication. It may, however, be a precursor of the exophthalmic disease.

Modern research has plainly demonstrated that goiter can be produced by a number of different toxic agents, inorganic or organic. In many goiter districts the drinking water has been shown to be responsible. Residuum of filtered water from certain fountains in Switzerland was found, when added to harmless water, to convey to it goiter-producing properties. Again, limestone districts show a large proportion of goitrous inhabitants. According to Kocher, "goiter water differs from goiter-free water in containing many more micro-organisms." McCarrison and others have recently afforded strong practical support to the bacteriogenic view of goiter. Animals allowed by McCarrison to drink only water contaminated with feces very readily developed goiter, and soil deposits on the sides and bottom of water channels, tanks, wells, etc., were found by him capable of contaminating the contained water. The participation of calcium in the etiology is explained on the ground that it affords, in the soil, a favorable medium for the pathogenic micro-organism responsible.

Bacterial infection from the tonsils can undoubtedly also provoke goiter; likewise infection from the teeth and gums, the nasopharynx, and many other situations.

That intoxication by intermediate protein waste products may provoke goiter is indicated by many clinical and some experimental observations.

Throughout, the effects of infections or other toxic materials on the gland are most easily understood when the view of the senior writer that the thyroid and parathyroids are intimately related to the defense of the body against poisons, exogenous as well as endogenous, is borne in mind. The organ becomes enlarged because it is the seat of an excessive defensive reaction against noxa circulating in the system.

Five distinct types of goiter may conveniently be recognized: (1) *simple hypothyroid non-toxic goiter* (simple or parenchymatous goiter), in which the increased functional demand has provoked congestion, hyperplasia, and enlargement of the thyroid. (2) *hyperthyroid or toxic goiter*, in which excessive secretory activity resulting from the hyperplasia has provoked the characteristic symptoms of hyperthyroidia or

even Graves's disease, though in most cases without exophthalmos; (3) *hypothyroid degenerative goiter*, comprising the colloid, cystic, and fibrous types, etc., in which the changes noted occur probably as complications of the hypothyroid non-toxic goiter; (4) *malignant goiter*; and (5) *congenital goiter* or *goiter of the newborn*, usually corresponding pathologically with the congestive or hyperplastic type of adults, and occurring frequently in the offspring of goitrous parents, and as a result of pressure during birth.

**Simple Hypothyroid Non-toxic Goiter.** The enlargement of the thyroid in this type of goiter is the expression of an effort on the part of a gland weakened through hereditary influence or previous local lesions to measure up, in times of stress, to the functional output required of it. Such a goiter rarely shows a true hyperplasia of the secreting epithelium, the chief changes being a pronounced hyperemia and an increase of the normal cellular elements.

The goiter tends to persist, unless the source of the functional stress on it can be found and removed, and tends to undergo colloid, cystic, or other changes. It tends, moreover, toward the production of cretinism or myxedema, and in many instances, careful observation will reveal some symptom or other of hypothyroidism, *e.g.*, bradycardia, hyperidrosis of the extremities, slight hypothermia and a tendency to cold feet and hands, and a reduction in urea excretion. Rheumatoid pains, often about the nucha or between the shoulders, and especially marked when the patient is in bed, are frequently observed. At first the gland enlargement may be noticed only upon palpation or by inspection during swallowing, coughing, or deep breathing. As the gland enlarges further, the trachea begins to be compressed or distorted, especially when one side of the thyroid is much larger than the other, causing dyspnea. Headache and dizziness from pressure upon neck-vessels may also occur.

An *x-ray* examination is indicated to ascertain whether an intrathoracic goiter or an enlarged thymus coexists.

**Treatment.** Goiters occurring in young subjects or pregnant women occasionally recede spontaneously when the causative factor has ceased to operate, but as a rule the enlargement tends to persist and become gradually more

marked, if left untreated. In endemic goiter, a change of drinking and cooking water is often an important measure in the treatment. A meat-free diet, or at least the omission of red meats from the food, is helpful in most cases. Often enterogenous autointoxication is an important factor, and wherever intestinal action is sluggish, saline aperients should be employed, especially sodium phosphate, the latter in 8-gram (2 dr.) daily doses. Certain Swiss observers, mindful of the possible water-borne origin, have administered intestinal antiseptics such as phenyl salicylate and betanaphthol, with notably beneficial results. Creosote carbonate, thymol, and zinc phenolsulphonate have also been used for bowel antiseptics. McCarrison,<sup>119</sup> attributing endemic goiter in India to the combined action of an ameba and of bacteria in the intestine—the thyroid being unable to overcome the combined toxic effects without undergoing enlargement—administered a mixed bacterial vaccine in 33 cases of parenchymatous goiter, with excellent results, one of the two sources of burden placed on the thyroid—the bacterial toxins—being thus eliminated. Langmead,<sup>120</sup> in 8 cases of parenchymatous goiter, gave vaccines of coliform bacilli obtained from the patient's own bowel, this causing the goiter to disappear in one case, and reducing it in the others.

As in exophthalmic goiter, nearby or remote foci of infection which might be factors in the thyroid enlargement should be diligently sought and removed. Catarrhal disorders of the nasal cavities, ears, tonsils, lingual tonsil, etc., are frequent causes requiring correction either by antiseptic measures, cauterization, or excision. In the young, adenoids may be a cause of goiter. Ulcerative pelvic conditions at times require attention.

Direct relief for the overworked thyroid gland itself is possible, this group of cases tending toward the hypothyroid type, by the administration of iodine or iodids, avoiding, however, cases with pressure-symptoms or rapid growth of the goiter. Sodium iodid, for example, may be given in 0.3-gram (5-gr.) doses three times a day, gradually increased, if well borne, to 0.6 gram (10 gr.). The drug should be given immediately after meals in a small tumblerful of water. Some cases respond better to Lugol's solution in 3- to 5- drop doses. In general,

iodin is safer than thyroid substance in these cases. Further to promote the action of the iodin, a 5 to 10 per cent. ointment of iodopetrogen (N. F.) may be rubbed into the gland daily, using a piece the size of a small hazelnut, until skin irritation begins to appear. The inunction may with advantage be preceded by a ten-minute downward and outward massage of the thyroid, the rubbing movements being synchronous with deep respirations. This procedure tends reflexly to cause contraction of the vascular channels in the thyroid.

A rapid pulse does not necessarily preclude the iodin treatment, unless accompanied by other symptoms of larval exophthalmic goiter. Where distinct cardiac adynamia is detected, especially when manifest in dilatation of the right ventricle, digitalin, 0.006 gram ( $\frac{1}{10}$  gr.) twice daily will assist, not only by improving circulation in the body at large, but likewise, in doing so, by relieving congestion of the thyroid.

As an adjuvant to internal treatment, electricity is apparently sometimes of value, presumably by promoting contraction of vessels. The galvanic current to the amount of 10 or more milliampères may thus be used on alternate days, the electrodes being placed on either side of the goiter. Haslebacher<sup>121</sup> has had good results in goiter from vibratory massage, and has also used the quartz lamp in 20 cases. Under such treatment, he reports, the symptoms of stenosis soon subsided, and after 2 or 3 exposures the gland became softer.

Surgical treatment will be referred to below under Hypothyroid Degenerative Goiter.

**Hyperthyroid or Toxic Goiter.** This group comprises those cases termed by Plummer "toxic non-hyperplastic goiter," in which, although some symptoms of hyperthyroidia are present, the typical hyperplastic glandular changes of Graves's disease do not exist. Clinically, most if not all of these cases strongly resemble instances of larval exophthalmic goiter. Such hyperthyroidia develops, according to Brenizer, in 20 to 25 per cent. of all cases of simple goiter, most frequently in instances of diffuse or encapsulated adenoma of several years' standing.

*Treatment.* Fowler's solution, 0.12 to 0.2 mils (2 to 3 m.) three times daily in a half-glassful of water, appears to be of value when a goiter tends to assume the Graves type. Bro-

mids and cold compresses over the gland are also likely to be serviceable. Where, however, symptoms such as tremor and tachycardia persist in spite of such treatment, the treatment for exophthalmic goiter (*q.v.*) should be resorted to. Among other measures, injections of quinin and urea hydrochlorid have proven highly effectual in the hands of L. F. Watson<sup>122</sup> in overcoming hyperthyroid symptoms, though not in removing the gland enlargement. He finds small infiltrations, frequently repeated, to be best. To avoid all chance of inducing acute hyperthyroid symptoms with the earlier injections, Watson makes preliminary injections of a few minims of sterile saline solution at 1 to 3 days' intervals, followed by injections of sterile water. Cases responding best are those of beginning hyperthyroidism. Where such treatment fails, thyroidectomy, or at least ligation of some of the thyroid arteries, may be resorted to.

**Hypothyroid Degenerative Goiter.** This type of goiter often results from the occurrence of some form of degeneration in a simple, parenchymatous enlargement. The degenerative change may be limited to one or several restricted portions of the gland, thus causing it to present, in most instances, an irregular or nodular surface. The latter peculiarity is thus the chief feature clinically distinguishing the degenerated from the simple, parenchymatous goiter. Again, in the latter, pressure from the outside reduces the size of the enlargement, causing vascular depletion, while nodular goiters yield but little to pressure. In large colloid or cystic growths, fluctuation may sometimes be discerned. Pressure-symptoms, due to nodular formations enclosed in the goiter, are far more likely to exist in the degenerative than in simple goiters, such symptoms comprising hoarseness, dyspnea, paralysis of the vocal cords, cyanosis, paralysis of various muscles of the arm, numbness of the fingers, etc. Myxedematous symptoms may be noted when fibrous or other retrogressive changes have been sufficient to cut down almost completely the functional activity of the gland.

Among the varieties of degenerative goiter are the *colloid*, *cystic*, and *fibrous* forms. With this group may likewise conveniently be mentioned *intrathoracic* and *constrictive* goiters, *lingual* goiter, and *hemorrhagic* goiter.



*Treatment.* As in other forms of goiter, detection and removal of a causative intoxication is essential, though since the degenerative processes mentioned are often later changes in the enlarged gland, complete results are not as frequently to be expected as in simple, parenchymatous goiter. Iodin or thyroid substance may in the colloid form give rise to untoward effects, but in most of the other types referred to may be administered, carefully and tentatively. Cases of long standing, even though the goiter be small, are those most likely to react unfavorably to iodine. In the cystic goiters, aspiration of the cysts may greatly hasten reduction of the gland.

Surgical treatment of the simple, parenchymatous type of goiter, or of the degenerative types, is indicated where, upon fair trial of non-operative measures, no results are obtained. In general, the following specific indications for operation are recognized: (1) disfigurement, where the growth is large; (2) symptoms from pressure on the trachea, esophagus, larynx, or other structures in the neck or upper thorax; (3) rapid enlargement, suggesting a malignant nature; (4) symptoms of hyperthyroidism, if not relieved by boiling water or quinin and urea injections; (5) infection of the goiter.

Often a definite indication for operation consists rather in some special location of the simple or degenerated goiter-tissue than in the size of the enlargement as a whole. At times a swelling not exceeding a cherry in size may cause urgent discomfort. Especially in intrathoracic goiter with pressure-symptoms—difficult to recognize unless the *x*-rays be used—is early intervention indicated. Colloid and adenomatous goiters, usually but slightly responsive to medical treatment, require operation in the presence of pressure-symptoms, the diseased part of the gland being removed and its better portions preserved. In the simple goiter of adolescents, which tends to subside later in life, operation is seldom indicated.

Nodular or cystic goiters, or goiters beginning to adhere to neighboring structures, demand operation, according to most surgeons. Where both lobes are enlarged, the larger and, especially, the deeper lobe is the one to be removed. After a unilateral operation the remaining lobe, it is said, will later

undergo a reduction in size. If a bilateral enlargement is symmetrical, however, C. H. Mayo<sup>123</sup> favors division of the isthmus, with double resection of the gland, as being indicated for the best cosmetic results. As E. H. Pool<sup>124</sup> points out, it is advantageous, though not imperative, to leave *in situ* the posterior portions of both lateral lobes, in relation with each of which a recurrent laryngeal nerve and two parathyroids usually lie.

Some thyroid nodules, *e.g.*, adenomata, are encapsulated, and can be removed by perforation of the gland substance and enucleation of the nodule with the finger or a blunt instrument. Resection-enucleation, applicable in large adenomata, consists in excising the thin layer of thyroid tissue over the tumor along with the latter, and then suturing the cut edges of the gland. In rapidly growing parenchymatous goiter in young subjects, arterial ligation has been advised.

In operations for uncomplicated simple goiter the mortality is only a fraction of 1 per cent. Operation is safest where the growth is rounded and even in outline—typically in cystic goiter. Risk is greater, however, where continued pressure on the trachea has resulted in bronchitis, emphysema, poor oxygenation, and impaired heart-action. Diffuse follicular colloid degeneration is also an unfavorable condition, the proportion of normal gland-tissue having been greatly reduced. In large, nodulated goiters, pressing on the trachea and only slightly movable, vascular ligation followed by unilateral excision is deemed the safest procedure.

**Malignant Goiter.** Malignant goiter usually occurs as a complication of simple goiter of long standing, but occasionally is primary. Sarcoma is much less common than carcinoma. Pain in an uninflamed goiter suggests malignancy, especially if a cachectic facies coexists. The lymphatics are involved early, as a rule, and the metastases show a predilection for the bones. Carcinoma of the thyroid is usually nodular; sarcoma, smooth. Occasionally accessory thyroid glands, situated between the trachea and esophagus or behind the latter, develop malignancy.

**Treatment.** Early operation is indicated, the whole gland being removed, but its capsule preserved whenever possible. In late malignancy with lymphatic involvement, operation is

hardly to be recommended, except for the relief of pressure symptoms, as it may accelerate the growth of the tumor.

**Congenital Goiter.** Diethlin, among 2292 cases of goiter, met with no less than 25 cases during the first year of life, and other authors have reported a much larger ratio. In some infants the goiter is purely congestive, being due presumably to pressure during parturition. In many more instances, however, it is of the parenchymatous type, and the child is the offspring of a goitrous mother. McCarrison,<sup>125</sup> from experimental work, has become convinced that congenital goiter is due to the effects of toxic substances derived from the maternal intestine upon the fetal thyroid.

In a goitrous infant sudden death may occur soon after birth. At times the clinical signs, with the exception, perhaps, of a slight swelling at the front of the neck, do not appear until several weeks or more after birth. The symptoms are practically those observed in adults. Congestive goiter from pressure may disappear permanently within twenty-four hours, or reappear intermittently.

*Treatment.* The various forms of artificial respiration, together with the use of oxygen, are helpful. If dyspnea persists, section of the isthmus, or exothyropexy, will give immediate relief. Tracheotomy is contraindicated, though intubation has been employed. Thyroid substance or sodium iodid given to the mother may at times lead to disappearance of the goiter in both mother and child. In the congestive form, cold compresses should be applied to the neck, and warm foot-baths or hot baths administered.

## DISEASES OF THE PARATHYROIDS.

The parathyroid glandules develop in pairs from the third and fourth branchial clefts on either side of the body, and are thus typically four in number—two superior and two inferior. In some human cadavers, however, even careful study of serial sections has at times failed to reveal more than two or three of the glandules. The superior parathyroids generally lie behind the middle third of the thyroid, at the level of the lower border of the cricoid cartilage; the inferior, behind the lower third of the thyroid. At times an inferior parathyroid

is situated at, or even below, the lower pole of the thyroid. Only rarely is a parathyroid embedded in thyroid tissue; usually, they are surrounded by fibers of the thyroid capsule, and cling to the latter when it is stripped from the thyroid. Small supernumerary parathyroids have been noted, generally below the thyroid, or within the thymus or the thyroid itself.

Each parathyroid is constituted of a compact mass of epithelial cells, together with a reticular stroma. In occasional instances the epithelia are formed into lobules, in the center of which a lumen filled with colloid may be found. The epithelia are divided into two types, the more numerous principal cells and a minority of the oxyphile or granular cells. The latter, it has been suggested, are functioning cells, and the clear elements, cells in a resting state. As regards proteid contents, Beebe<sup>126</sup> notes that, whereas the thyroid contains an especially large amount of globulin, the parathyroids contain chiefly nucleoproteid.

The blood supply of the parathyroids is derived chiefly from the inferior, less from the superior thyroid arteries. Anastomoses may also bring blood from the tracheal, esophageal, and pharyngeal vessels. Nerve fibers ending in immediate relationship with the vessels to the parathyroids have been shown to exist by Rhinehart.

Whereas removal of the thyroid alone is followed by a prolonged post-operative life, thyroparathyroidectomy, or parathyroidectomy alone, results in severe nervous symptoms—tetany—soon terminating in death. The morbid effects, characterized especially by a tendency to spasms or convulsions, usually begin about twenty-four hours after removal of all the parathyroids, death generally following in three to five days. In dogs, a single parathyroid suffices in most instances to obviate tetany, but in some animals even removal of only two parathyroids out of four has been observed to induce the disease.

Functionally, according to some observers, the parathyroids operate independently of the thyroid. This view is suggested by the differences in the effects of removal of the two types of tissue. Various facts, however, tend to show that the independence of the two organs is not as complete as might be supposed. Thus, some evidence has been collected to the

effect that after thyroidectomy the parathyroids may assume in some degree the functions of the thyroid. As shown by Vassale, moreover, injection of thyroid extract is capable, like parathyroid extract, of allaying the convulsive disorders which follow complete parathyroidectomy. According to Edmunds, extirpation of the parathyroids causes hypertrophic histological changes in the thyroid. Vassale and Generali<sup>127</sup> found that after death from parathyroidectomy the thyroid contains no colloid, thus suggesting that the formation of the thyroid product is in some way related to the functions of the parathyroids. In experiments performed by Gley,<sup>128</sup> an apparently complete parathyroidectomy, with partial thyroidectomy, did not prove fatal until the remaining lobe of the thyroid was likewise removed.

Again, it has been asserted that grafted pure thyroid tissue is capable of assuming the functions of both the thyroid and the parathyroids, thus arresting the convulsive disorders caused by parathyroidectomy, and preventing death. Tanberg<sup>129</sup> noted that if insufficiency of the parathyroids was induced in meat-fed animals by extirpation of two or more of the glandules, the usual hypertrophy of the thyroid due to the meat diet did not develop. Further, thyroid hypertrophy induced by a meat diet disappeared after excision of a sufficient number of parathyroids. This, according to Tanberg, points to some interrelation between the thyroid and parathyroid glands, at least to the extent that parathyroid insufficiency interferes with the function of the thyroid. Gley's conception of the relationship of the two structures recognizes a functional association in the sense that one organ serves to complete the work of the other. According to Sajous, Sr.,<sup>130</sup> the secretion of the parathyroids, passing into the lymph spaces with that of the thyroid, eventually reaches the heart and general bloodstream, and, like the thyroid product, becomes a constituent of the hemoglobin. The combined thyroparathyroid products thereupon serve to enhance general oxidation "by increasing, as a ferment, the vulnerability of the phosphorus, which all cells, particularly their nuclei, contain, to oxidation by the adrenoxidase in the blood." The parathyroid secretion is also deemed by him to co-operate with the thyroid secretion in increasing the germicidal and antitoxic properties of the blood.



Removal of the parathyroids was found by Jeandelize,<sup>131</sup> like thyroidectomy, to lower the body temperature. That the parathyroids, moreover, are in a sense, like the thyroid, protective and antitoxic organs is indicated by the effects of their removal, the resulting convulsive phenomena strongly suggesting a capacity on the part of the parathyroids to destroy or prevent the morbid action of certain spasmogenic poisons. Further evidence in favor of such a conception is afforded by the fact that the blood and urine of parathyroidectomized dogs have been found by Rogowitsch and others to cause convulsions in normal animals. The observation that injection of parathyroid extract will arrest, at least temporarily, the convulsive phenomena of parathyroidectomy has generally been accepted as proof that some antitoxic substance is contributed to the blood by the parathyroids. MacCallum and Voegtlin<sup>132</sup> having found that calcium salts arrest tetany due to parathyroidectomy, the antitoxic action of the parathyroids has been ascribed to some influence exerted by them on calcium metabolism. This question will be further taken up under the heading of Parathyroid Insufficiency. According to Kendall,<sup>133</sup> who, as we have seen under Diseases of the Thyroid, has advanced the hypothesis that the function of the thyroid is to regulate the deaminization of amino-acids, the parathyroid function is to detoxicate the ammonium carbonate formed by union of the removed amino-group with the carbon dioxid in the blood. He states that, from the results of previous investigators, it seems probable that ammonium carbonate is responsible for the tetany following parathyroidectomy.

No group of phenomena characteristic of excessive parathyroid activity being as yet known, consideration of the morbid conditions of the parathyroids comprises chiefly a discussion of tetany or other manifestations of parathyroid insufficiency and of tumors of the parathyroids.

### PARATHYROID INSUFFICIENCY (HYPO-PARATHYROIDIA).

**Postoperative Tetany.** The convulsive phenomena following complete parathyroidectomy range from a mere tendency to spasm to violent tetanic or epileptoid paroxysms, with

foaming at the mouth, and finally death from "cramp asphyxia." Fibrillary tremors are also a marked feature, and, as in strychnin poisoning, the convulsive phenomena are easily initiated by even mild peripheral sensory stimuli. The temperature rises during the convulsive paroxysms, but falls considerably below normal during the intervals. Oxygenation is plainly impaired, and the general condition is one of weakness and, as a rule, somnolence. Pruritus is a manifest symptom. The heart-beat is rapid, except after convulsions, and the respirations are greatly accelerated. Death occurs more frequently during the state of depression following a period of hyperexcitability and convulsions than during an attack of hyperexcitability.

In man, tetany has at times resulted from accidental removal of an excessive amount of parathyroid tissue during thyroidectomy. As a rule, beginning trismus and facial twitchings and tingling are the initial symptoms. The extremities are then affected, the hands assuming the "main-en-griffe" or "obstetric" positions, the forearms becoming flexed, and the feet being cramped, often in the equinovarus position. In severe cases, opisthotonos may develop, respiration become difficult, and the circulation impaired. As a rule, the first signs occur on the third or fourth day after the operation. The paroxysms vary in frequency from one to many a day. Where the parathyroids have been merely injured during the operation, tetany ceases as repair takes place.

Tetany, usually in a milder form, is also met with clinically as a result of various lesions or intoxications of the parathyroids. These non-operative forms of hypoparathyroid tetany will be referred to under the heading Organic and Functional Disorders of the Parathyroids.

During tetany following removal of the parathyroids the irritability of the peripheral nerves and, apparently, of all other nervous tissue to the galvanic current is greatly increased. Intravenous injection of soluble salts of calcium having been shown almost instantly to remove this excessive irritability, MacCallum and Voegtlin<sup>134</sup> were led to formulate the hypothesis that the function of the parathyroids is in some way to control calcium metabolism, and that after parathyroidectomy the body fluids and soft tissues are deprived of soluble cal-

cium, this, in turn, resulting in the abnormal nervous irritability of tetany. The observation of D. W. Wilson, Stearns, and their co-workers,<sup>135</sup> however, that parathyroidectomy is soon followed by an alkalosis, and the facts that hydrochloric acid is equal in therapeutic power to calcium in experimental tetany, and that continuous administration of calcium is not capable of preventing death permanently after parathyroidectomy, are complicating factors, which led Voegtlin<sup>136</sup> to recognize the probability that the parathyroids have other functions besides their influence on calcium metabolism, and the possibility that tetany is but a partial expression of the metabolic disturbances induced by removal of these organs. Kendall's<sup>137</sup> view of the causation of tetany has already been mentioned.

Koch<sup>138</sup> asserts that after parathyroidectomy, methyl cyanamide accumulates in the body, and is responsible for the death of the animals.

*Treatment.* Calcium lactate was found of some value in tetany by MacCallum and Voegtlin. It should be given in full dosage, at least 0.6 gram (10 gr.) every hour or two, or by rectum in larger doses in saline solution. Absorption of calcium salts from the alimentary tract being, however, slow at best, while subcutaneous and intramuscular injections of such salts cause marked irritation, intravenous use is by far the best procedure in emergency cases. Winternitz,<sup>139</sup> for example, thus gave 100 mils ( $3\frac{1}{3}$  f $\bar{5}$ ) of a 4 per cent. solution in a non-operative case of tetany, with satisfactory results. Marked benefit from calcium treatment lasts, however, but about twenty-four hours, or somewhat longer, after which the tetany symptoms begin to return, growing progressively worse thereafter.

Benefit from the oral use of parathyroid extracts appears to be only slight, MacCallum finding that large quantities were necessary to give any result. Much more effective is the subcutaneous use of an extract of fresh parathyroids. Brantham,<sup>140</sup> in preparing such an extract, placed 5 fresh beef parathyroids in 1:1000 mercuric chlorid solution for ten minutes, then cut them into pieces aseptically, and ground them in a mortar with 400 mils (13 oz.) of sterile saline solution. The product was finally filtered through gauze, and infused under the patient's breast.

As with calcium, relief from parathyroid extract is only evanescent. Transplantation of parathyroid tissue suggests itself as a possible means of avoiding this. While Halsted found, however, that autotransplantation of parathyroids is feasible in the presence of hypoparathyroidia, transplantation from other individuals has apparently never been thoroughly successful, the implant causing improvement for, *e.g.*, a couple of weeks (a result sometimes sufficient to save life by tiding a case over a critical period of parathyroid deficiency), then becoming absorbed.

In the diet, in the presence of hypoparathyroidia, it is advantageous to exclude meats and other substances rich in nucleins, to minimize the formation of spasmogenic wastes. Water should be taken freely for eliminatory purposes. Complete rest tends to reduce the number and severity of the paroxysms.

#### **Organic and Functional Disorders of the Parathyroids.**

Mild forms of tetany sometimes result from partial lesions of the parathyroids, such as tuberculosis, interstitial hemorrhage, etc. Chvostek's sign (facial spasm upon tapping the facial nerve), Hoffmann's sign (hyperesthesia upon percussion of sensory nerves), etc., are clinically evidences of the existence of tetany. Infectious diseases may be primary causes of tetany through injury to the parathyroids. Tetany is probably also at times a result of excessive demand upon and exhaustion of the parathyroid function, or of inhibition of the activity of the glandules through the action of circulating toxic materials upon them. Whether tetany accompanying gastro-intestinal disturbances, uremia, and violent excitement or exertion may be due to parathyroid insufficiency is not as yet definitely established. That tetany during pregnancy and lactation (usually the former) may be due to excessive functional demand upon the parathyroids is suggested by the observation of Vassale and Generali, and many others, that after partial removal of the parathyroids tetany during pregnancy will not infrequently follow. Parathyroid disease has been thought a factor in the pathogenesis of paralysis agitans, in which lesions of the glandules have sometimes been found and considerable improvement procured by parathyroid therapy. Harbitz<sup>141</sup> has reported 2 cases of tumors of the parathyroid, 1 associated

with osteomalacia, and the other with paralysis agitans. He states that of the few cases of parathyroid tumor recorded, few have presented symptoms bearing on the functions of these glands.

*Treatment.* In the presence of signs of parathyroid insufficiency, the measures described under Postoperative Tetany are indicated. In paralysis agitans, protracted benefit has been reported by Berkeley<sup>142</sup> from the administration of parathyroid nucleoproteid, prepared by the Beebe process, in doses of 20 drops a day. Dried parathyroids in the daily dosage of 5 to 7 capsules, each equivalent to 0.03 gram ( $\frac{1}{2}$  gr.) of the fresh glandules, may also be used.

## DISEASES OF THE THYMUS.

The thymus gland was shown by Hammar<sup>143</sup> to increase in size from birth to puberty, at which time its average weight is 25 grams. It then diminishes in size, at first rapidly, weighing 5 grams at the twenty-fifth year, then more slowly to 50 or 65 years, when it may weigh but 0.73 gram. Even in old age, however, it usually retains small remnants of parenchyma. Wide and rapid fluctuations in the weight of the thymus may occur at any time through starvation, exhaustion, or wasting diseases.

Histologically the thymus is deemed primarily an epithelial structure invaded by lymphocytes from neighboring mesenchyme, these lymphocytes then so proliferating as to constitute the main mass of thymus tissue, through which course as a reticulum the epithelial cells. Careful study has shown that the thymic lymphocytes are very similar, if not identical, with those found in lymph glands. Biedl<sup>144</sup> recognizes a difference, however, in that the amount of nucleinates is at least five times as large in the thymus as in the lymph glands. The thymus is thus apparently an organ capable of supplying nucleins, bodies which are rich in phosphorus, to the body at large. "The more one studies the thymus," states Pappenheimer,<sup>145</sup> "the more certain becomes the conviction that the constant, and under some conditions, excessive disintegration of nuclear material is the most obvious form of activity which takes place in this organ."



That deficient thymus activity might react, during the period of growth, upon the various structures of the body most rich in phosphorus-containing material is a permissible supposition in view of the above facts. Huiskamp<sup>146</sup> found nucleohiston, which contains no less than 3.7 per cent. of phosphorus, to be the most abundant proteid in the thymus. Examining the results of experimental thymectomy in the hands of various experimenters, one notes that the changes observed as a result of this operation in many instances correspond rather closely with those to be expected from lack of phosphorus distribution in the system.

The labors of Basch (1906), Klose and Vogt (1910), Lampé (1913), and others seemed, until recently, to have definitely shown that, *e.g.*, in dogs completely thymectomized in early life, bone deformities of a rachitic type appear about the fourth month. The paws become curved inward, and the cranium is stated to be large, flat, and short. Toward the fifth month the animal becomes somnolent, depressed, and cachectic, losing weight until, between twelve and eighteen months later, coma and death supervene. More recently, Nordmann,<sup>147</sup> and Howland, McClure and Park,<sup>148</sup> in experiments which appear to have been performed with great care, have failed to reproduce these results, and Pappenheimer<sup>149</sup> has been led to conclude that loss of the thymus in young animals is not of prime importance, but is readily compensated for in ways not yet understood.

The large number of previous positive results seem sufficiently conclusive for the present. The possibility suggests itself, moreover, that the experimental discrepancies may have been due to different feeding methods in different series of experiments; were the diet relatively poor in phosphorus, the thymus deficiency might lead to distinct manifestations where a richer diet would cause no disturbances whatever. If the thymus is considered, as is manifestly the case, only one of a number of reservoirs or factories for phosphorus-containing bodies, it can readily be understood that under different circumstances the thymus deficiency might vary enormously. Were enough of these bodies for existing needs stored or elaborated elsewhere, thymectomy might be entirely devoid of effect, at least in relation to the osseous system.

In the human subject in particular, certain findings seem to suggest a relationship between thymus deficiency and the production of idiocy. At Bicêtre Hospital, according to Morel,<sup>150</sup> 75 per cent. of 408 non-myxedematous idiotic children examined *post-mortem* showed absence of the thymus. In 28 mentally weak children examined by Bourneville, the thymus was likewise absent. Basch, Klose and Vogt, Morel and others observed marked mental impairment in puppies in the fifth or sixth month after thymectomy. A possible influence of the thymus on the growth of the body as a whole is indicated by Gudernatsch's<sup>151</sup> observation that feeding thymus substance to tadpoles greatly prolongs their early growth period, the tadpoles becoming unduly large, but metamorphosis into frogs being, on the other hand, correspondingly delayed. R. W. Wilcox<sup>152</sup> claims to have obtained a gain in height of 9½ inches in an undersized boy by administration of thymus. According to some, thymectomy causes delayed and defective development of the reproductive organs. The experiments of various observers on this point have, however, been very contradictory.

On the whole, as a provisional conclusion, it may be stated that there is considerable evidence at hand tending to confirm the view advanced by Sajous, Sr.,<sup>153</sup> that the function of the thymus is to supply, through the agency of its lymphocytes, the excess of phosphorus in organic combination which the body requires during the developmental period.

### THYMUS INSUFFICIENCY (HYPOTHYMIA).

Under various conditions, such as acute or chronic inanition, infectious processes, and the influence of the *x*-rays, a premature involution of the thymus may occur. Pappenheimer<sup>154</sup> speaks of a massive destruction of the thymus lymphocytes as occurring under such conditions, and remarks on the "extraordinary fragility" of these cells, which leads to a "dissolution of nuclear material *en masse*." Various experimenters have recorded an unusual susceptibility of the body to infection after thymectomy, Hart and Nordmann<sup>155</sup> recognizing that the organ takes an active part in the resistance of the organism to infection. Especially important, according to

Sajous, Sr.,<sup>156</sup> is the relationship of thymus insufficiency to certain forms of deficient mental development, in particular Mongolian idiocy.

**Mongolian Idiocy.** In this condition are combined the typically Mongolian slant of the eyes and prominent epicanthal folds, with various defects in bone development resembling those reported in thymectomy experiments in animals. The stature is low—the long bones, particularly those of the legs, being abnormally short; the chest is flat; the nose squatty, the ears, small and undeveloped; and the hands stubby and square, with the fingers short, but tapering. The head as a whole appears unusually rounded, and is small in size, and further evidence of defective bone nutrition is manifest in the frequency of rickets, of which distinct signs are, as a rule, noticeable at birth. Palatal deformities exist in about two-thirds of the cases. Appearance of the teeth of both dentitions is delayed; the teeth are irregular, and soon undergo caries.

Whereas the bony conditions just mentioned suggest deficiency of the thymus, the dry and rough skin of certain cases, and the thick, heavy, protruding tongue, seem indications of hypothyroidia. Hypothermia, with marked sensitiveness to cold and sluggish circulation, is also a significant feature. Relaxation of the general musculature, voluntary and involuntary, as well as of the ligaments, is commonly noted. The relative insufficiency of the thymus and thyroid glands, and possibly also of the adrenals, in this disease render the Mongolian subjects peculiarly susceptible to infections. As a matter of fact, respiratory and intestinal infections form such an obstacle to continued life in these cases that the twenty-fifth year is reached in but 9.4 per cent. of them (Wiggandt).

Deficiency in the nucleins normally supplied by the thymus may account for the extremely slow mental development of the typical Mongolian idiot. Abnormal quiet and greatly postponed appearance of the powers of observation are typical throughout, though these children are apt to be amiable in temper, and show a marked predilection for ape-like mimicry as well as for music. Leeper,<sup>157</sup> in a study of 176 Mongolian idiots, found no less than one-half of them to be the last-born of large families. Again, these children occur as the offspring

of aged couples, or where a marked disparity in age exists between the parents. To use a homely comparison, the factory being worn out *in toto* or in part, the product is below par. Sajous, Sr.,<sup>158</sup> has made the suggestion that the peculiar Mongolian facies of these children may not be due to mere hazard, but may reproduce, to some extent, effects resulting in the Mongolian branch of the human family from subsistence through many generations on an "unbalanced" diet. A diet consisting too exclusively of overmilled rice is deficient in phosphorus-containing compounds or vitamins, and on this account, underdevelopment of the tissues in which phosphorus containing compounds are prominent, viz., the osseous and cerebrospinal systems, may logically be expected. Common to the Eastern Asiatic races and the Mongolian idiot are slanting eyes, narrow palpebral fissures, marked epicanthus, high cheek-bones, a yellowish and doughy skin, straight hair, a squatty nose, and, lastly, low resisting-power to infectious diseases, especially in the rice-fed "coolies." These features are less constant in the upper classes of Asiatics than the lower, presumably owing to the greater variety of food ingested by the former, which obviates the deficiency of phosphorus intake.

The pathology of the nervous system in Mongolian idiocy shows merely an imperfect cellular and general development of the brain, without any definite organic lesion. To this are added certain nutritional lesions in the osseous system, already referred to. As regards the thymus itself, percussion and radiography indicate absence or atrophy of this organ in some and hypertrophy in other cases, the latter condition probably representing an effort at compensation in a partly diseased gland, as in compensatory hyperplasia of the thyroid in hypothyroid goiter.

*Treatment.* On the whole, the results of treatment in Mongolian idiocy have not been encouraging. Probably more could be done for these cases, however, than has hitherto been accomplished were the physician to be constantly on the watch for it where the underlying conditions exist, viz., aged parents, marked discrepancy in age of parents, markedly prolific parents, strong emotion or affliction in the mother, syphilis, or alcoholism. Unusual quiet in a baby, with loose joints and

persistent helplessness, is also a suggestive condition which should lead to careful scrutiny for facial characteristics of Mongolism. Treatment through the nursing mother may then be instituted, dried thymus gland, 0.3 gram (5 gr.), and dried thyroid and pituitary gland, 0.06 gram (1 gr.) of each, being given three times daily during meals, and an ample, varied diet prescribed. Where maternal or wet-nursing cannot be carried out, direct nursing using goat's milk in some such way as is employed in Italy, Egypt, and certain other countries, is appropriate, or, if cow's milk must be used, it should be fresh from the udder, being directly pumped into the nursing bottle before feeding.

Treatment of the older Mongolian child consists in insuring good nourishment and country air, as well as in giving organic remedies. The doses already mentioned for the mother are appropriate for a child of five years, but the dose of dried thymus may with advantage be gradually increased, up to 1 gram (15 gr.) three times a day. The dried thyroid and pituitary may likewise be increased if stigmata of disease of the corresponding gland are noticeable. Iron and syrup of hypophosphites may be of service as adjunct remedies. Where the tonsils are enlarged or adenoid tissue present, removal is indicated.

The mental condition may be further improved by persistent, systematic education, in the accomplishment of which the patient will, as a rule, readily co-operate.

**The Thymus in Backward Children.** Apart from actual idiocy, there are many instances of relatively slight impairment of the mental powers in which the ductless glands appear to play at least a partial rôle. The importance of the subject is shown in the observation, in the course of investigations conducted by the Russell Sage Foundation in thirty-one American cities, that over 20 per cent. of all school children belong in the "retarded" class. In these children an actual mental defect is not considered to exist, but the development of the mind is hindered by unfavorable environmental, dietetic, or other factors. Enlarged tonsils or adenoids, errors of refraction, insufficient food, absence of effort on the part of the parents to extend the vocabulary and develop understanding, removal of the stimulus attending association with other children, etc., are all



prejudicial influences which must be overcome before organotherapy can be expected to give results. Anemia, tuberculosis, or inherited syphilis must, where present, be overcome in so far as is possible.

The stigmata of deficiency of one or more of the ductless glands, in particular the thyroid, pituitary, adrenals, or thymus, must then be carefully sought and organic remedies administered according to the findings.

Where the thymus is deficient, which is often the case when larval myxedema is present, any existing mental torpor—illustrated in a tendency to answer questions slowly and with hesitation—is *ipso facto* increased, and the child's osseous system will be likely to show deformities suggesting rickets, together with the looseness of the ligaments already referred to under Mongolian idiocy. Sometimes skin disorders, especially a tendency to warts and eczema, coexist. In cases thus exhibiting hypothymeric stigmata, dried thymus gland 0.3 gram (5 gr.), gradually increased to 0.6 gram (10 gr.), and 0.03 gram ( $\frac{1}{2}$  gr.) of dried thyroids, taken in the course of each meal, together with 0.12 gram (2 gr.) of calcium lactate after meals, will usually afford marked improvement. In some cases the general health and mental condition improve in a parallel fashion; in others the mental deficiency persists even though the general health is greatly improved.

In all these cases of retarded mental development, a necessary feature in the treatment is carefully to adjust the work required of the child to his actual mental capacity at the time. To estimate accurately the degree of backwardness in a given case, the Binet-Simon method or one of its modifications is most appropriate. Special classes for backward children should be available in all communities. To such classes the children grouped according to the Binet-Simon test should be referred for the instruction appropriate to each. Aided and encouraged, but not goaded, by its teachers, the backward child will thus often become an object of surprise in the rapidity of the progress shown. If driven, on the other hand, beyond the working powers of its brain, it tends soon to become discouraged, unwilling, stubborn, and irritable, and later not infrequently to drift toward criminality, and find its way to a reformatory or prison.

### HYPERPLASIA OF THE THYMUS.

Abnormal size of the thymus may be a result either of retarded involution, the gland remaining distinctly larger than the average size indicated by the age of the patient, or of an actual hyperplasia, the organ growing to a larger size than normal. Enlargement of the thymus has repeatedly been observed to follow removal of both testes, and is also an acknowledged feature of many cases of exophthalmic goiter, the thymus often taking part, indeed, in the pathogenesis of the latter. Apart from these conditions, thymus enlargement is chiefly of clinical interest in relation to three forms of disturbances, viz., status thymico lymphaticus or status lymphaticus; thymic asthma, manifested as a more or less chronic dyspnea or in paroxysms of stridulous breathing, and thirdly, thymic death, the sudden exitus of apparently healthy subjects, usually infants, in the absence of pre-existing signs of compression of the trachea.

Status lymphaticus was first emphasized by Paltauf in 1889 as a condition characterized by hypertrophy of the entire lymphatic system, including the thymus, the cervical nodes, and the lymphatic tissues of the axilla, mesentery, tonsils, and spleen. To this syndrome were later added by Bartels the presence of a small heart, a narrow aorta, a large brain, degeneration of the thyroid, a small vagina and infantile uterus, and a general lowering of the resisting power of the organism to infection. The thymic hyperplasia in these cases may be general—a form found, according to George Dock,<sup>159</sup> probably only in infants—but usually is limited to the medulla of the thymus, the cells in this section of the organ being increased in number, while the cortex may be actually hypoplastic. As was recognized from the first by Paltauf, status lymphaticus predisposes to sudden death from causes which ordinarily would exert little or no harmful effect.

Direct diagnosis of the thymic enlargement is often not an easy task. Such obvious indications as a swelling above the manubrium sterni, prominence of the manubrium, and a tumor rising in the jugular fossa in inspiration or in crying, as Dock<sup>160</sup> has pointed out, are rarely present, the thymus often lying deeply in the thorax. The bulk of the enlarged gland

lies, as a rule, behind the upper part of the sternum, extending, however, more to the left of the midline than to the right. Occasionally the organ can thus be mapped out by gentle percussion, the area occupied by it forming usually a triangle, with its base at the level of the sternoclavicular articulation, and its blunt apex lower down, above the level of the third rib. If the dullness extends laterally over 1 centimeter (0.39 in.) beyond the margin of the sternum, morbid enlargement of the thymus is indicated. According to Jacobi, the dull area moves upward when the head is thrown far back. Among other indications of enlarged thymus, special stress has been laid upon hyperplasia of the lymphatic tissues at the root of the tongue. Often a thymic enlargement is discernible by examination with the *x*-rays; in some instances, however, such examination has proven misleading, no thymic swelling being found upon operating, in spite of positive *x*-ray indications. Lange, of Cincinnati, has worked out a careful technic of *x*-ray diagnosis, which is described by A. Friedlander.<sup>161</sup>

That an abnormally large thymus may at times cause pressure symptoms is doubted by no one, but there is still considerable difference of opinion as to the frequency with which such symptom-causing enlargement exists. The symptoms themselves, which develop suddenly or gradually, usually during the first year of life, and at times are witnessed immediately after birth, are in general manifestations of tracheal stenosis. The dyspnea may be constant or paroxysmal, and is usually made worse by crying, fits of anger, rapid backward bending of the head, or some acute infection, diphtheria in particular. Stress has been laid by various authors on a presumed special susceptibility of the thymus to rapid congestion, due to its arterial supply being more abundant than its efferent venous channels. Cyanosis is another typical symptom, which may be the result, however, not only of pressure upon the air passages, but also of compression of the vagus nerves, the great vessels, and the heart itself. The pulse-rate may be slowed, doubtless likewise through pressure on the vagus nerves. Voice abnormalities are ascribed either to tracheal pressure or to compression of the recurrent laryngeal nerve, and range from slight temporary hoarseness to aphonia. Inspiratory retraction of the supra- and infra- sternal regions,

in the absence of hoarseness, has, however, been considered a suggestive manifestation. Pressure on pulmonary vessels may actively promote congestion of the bronchial vascular distribution, and lead eventually to a capillary bronchitis, which in turn may pass into bronchopneumonia. In some cases, it is asserted, there is also pressure upon the esophagus, which may be such as completely to prevent swallowing.

In the "status thymicolymphaticus" the symptoms just mentioned are apt, according to F. H. Falls,<sup>162</sup> to be less marked, the predominating features being the general enlargement of lymphatic structures, a pasty skin, adiposity, and associated changes in the chromaffin system. In these cases especially is sudden death believed to occur. Opinions are still markedly at variance, however, as to the frequency, or even the possibility, of true "thymus death." According to Hammar,<sup>163</sup> investigation in 16 personal cases revealed no abnormalities of the thymus which would account for sudden death; this observer is disposed to ascribe the fatal ending to other ductless glands. Nordmann<sup>164</sup> ascribes thymic asthma not to mechanical pressure by, but to hyperfunction of, the thymus. Again, according to J. Grossman,<sup>165</sup> autopsies after "thymus death" have disclosed local anemia, flattening, partial obliteration, and atrophy of the tracheal wall, which have been proven due to compression of the trachea by an enlarged thymus. Fatal cases showing an apparently normal thymus are explained as having occurred through an edema or congestion of the gland, which disappeared after death. Schoeppler<sup>166</sup> asserts that in a case of sudden cyanosis and death in a child a year and a half old the thymus was found to weigh 85 grams, and the trachea was so compressed by it that a sound could be passed through only at the sides in the narrowed portion.

Jacobi is quoted by Falls<sup>167</sup> to the effect that pressure from acute congestion of the thymus on the trachea, great vessels, and nerves explains a certain number of cases of thymic death, but not all. Garrod<sup>168</sup> found there was evidence that not a few cases of supposed thymic death had actually been due to suffocative bronchopneumonia. Griffith<sup>169</sup> has expressed himself as believing that such deaths are cardiac deaths, that there is no anatomic proof of sudden thymus congestion, and that the most generally accepted theory accounts for the condition

as a neurosis, perhaps toxic in origin, sudden cardiac arrest occurring for some unknown cause which varies with the case. Gismondi<sup>170</sup> lays stress on direct compression by the thymus, not only of the trachea, but of the veins from the brain and arm, thus impeding indirectly the circulation in the veins of the already enlarged thymus itself, and setting up a vicious circle. Simultaneously the bronchial mucosa is congested, compression of the pulmonary veins interferes with blood aëration; through the combined action of these unfavorable conditions dangerous asphyxia may suddenly develop.

### TREATMENT.

Not infrequently spontaneous recovery from thymic asthma takes place, even where dyspneic seizures have recurred for several years (Rehn). Measures to accelerate involution of the gland are, however, available, and likewise precautions appropriate for warding off attacks of dyspnea or actual asphyxia. In young infants without especially alarming symptoms, Gismondi<sup>171</sup> found painting with iodine useful to hasten thymic retrogression. Particularly to be avoided for the prevention of seizures are excitement of the child, crying or screaming, running or jumping, swimming, baths at extreme temperatures, and the throwing of the head far backward. The child should live out of doors in an even climate, under hygienic conditions, and with a well regulated diet.

X-ray treatment of the enlarged thymus has been applied by a number of observers with successful results. Although some authors have held that such treatment should be restricted to older children and adults, more recent observations seem to have shown that it is attended with but little danger, even in young children. Friedlander<sup>172</sup> deems the x-ray treatment remarkably efficacious. In the average case, improvement of symptoms was noted within twenty-four to forty-eight hours. The Lange technic was used, a Coolidge tube and 9½ inch spark being employed, with the rays filtered through 4 millimeters of aluminum and a piece of thick leather. The target skin distance was 9 inches, and the routine exposure, 25 milliampereminutes. In mild cases a single dose over the anterior aspect of the chest sufficed, while in more urgent cases 50 milliampereminutes were administered, 25 anteriorly



and 25 posteriorly. The interval between treatments was usually one week unless urgent symptoms indicated more frequent applications. Stress is laid on sufficiency of dosage, failure to administer full doses and repeat them promptly having in very urgent cases led to fatalities under  $x$ -ray treatment. In a series of over 100 cases referred to by Friedlander there were 4 deaths.

According to P. H. Cook,<sup>173</sup> cases have been recorded in which symptoms were relieved in three and a half hours after application of the  $x$ -rays. The same writer quotes Lange as urging that recurrences due to regeneration of the gland should be kept under observation and controlled by further treatment. Lange advocates  $x$ -ray therapy as a precautionary measure in children of the "lymphatic type," to enable them to withstand intercurrent disease or anesthesia which would otherwise prove fatal. Gismondi<sup>174</sup> warns against a too violent  $x$ -ray treatment, reporting a case in which a mild tendency to rickets was whipped up into a severe grade of the disease by a series of excessive exposures. He would set the limit of a single dose at 5 or 6 H units, an aluminum filter being used.

Medication with dried thymus gland has been attempted in these cases, but without effect on the enlarged organ, as might have been expected. Good results from daily administration of adrenalin in small doses have, however, been reported by Franchetti and Pende. The latter observer prefers, moreover, a mixture of adrenalin with pituitary extract, ascribing to this combination a definite inhibitory property in relation to the enlarged thymus.

Surgical removal of a portion of the thymus has been carried out successfully in a relatively large number of cases, but the procedure is attended with a higher mortality than  $x$ -ray treatment. C. A. Parker,<sup>175</sup> analyzing 50 cases of thymectomy at all ages, noted 17 deaths, but reported a successful case of his own, and recommended surgical intervention when the thymus causes symptoms. Total thymectomy appears to have been found highly unsatisfactory, metabolic disturbances and rachitic phenomena following, at least in some instances. The usual procedure is therefore a partial intracapsular thymectomy. This may be performed by effecting an entrance into the thorax above the suprasternal notch. General or local anes-

thesia may be used, according to indications, the fact being borne in mind that in status lymphaticus with enlarged thymus death during general narcosis has been not infrequent. Falls<sup>176</sup> deems it doubtful whether thymectomy should be undertaken except where there is severe tracheal stenosis.

## DISEASES OF THE PITUITARY BODY.

This organ consists of three distinct parts, viz., the anterior or "glandular" lobe, constituting approximately three-fourths of the whole; the pars intermedia, a narrow, whitish layer situated behind the anterior lobe and constituting a lining around the posterior portion; and the posterior lobe itself, rounded in shape and partly enclosed by the larger anterior lobe. Histologically, the anterior pituitary exhibits two main types of epithelial cells, the chromophiles, which have a marked affinity for either basic or acid stains and embody granules apparently of secretory origin, and the chromophobes, which do not stain so readily and contain no clearly manifest granulations. These cells are disposed in solid columns, and between the latter course broad, thin-walled blood channels. The pars intermedia consists of several layers of finely granular cells, which produce a colloid material believed by some to contain the active principle or principles in pituitary extract.

The posterior or neural lobe, is coated posteriorly, according to Berkley,<sup>177</sup> by a layer of gray matter composed of ependymal cells three or four deep. The greater part of this lobe, however, is characterized by a rather dense aggregation of nerve-cells, more or less divided into subsidiary groups by connective tissue partitions carrying blood-vessels. These nerve-cells are of various types, some being different in structure from any other nerve-cells in the central or peripheral nervous systems. Berkley considers it doubtful whether any of the fibers from these cells pass out of the posterior lobe into the infundibulum or stalk of the pituitary, which connects the latter with the base of the brain proper. Ramon y Cajal, Andriezen, Gentès, and others, however, were able to satisfy themselves that nerve fibers do pass from the posterior lobe and the pars intermedia to the tuber cinereum and even beyond. Another feature of interest with regard to the hypo-

physis is the differing embryological derivation of its several sections, the anterior lobe and pars intermedia developing from the ectodermal epithelium of the primitive oral cavity, while the posterior lobe is derived from the embryonic brain or, more particularly, from the infundibulum, the latter attached to the floor of the third ventricle.

The composite make-up of the hypophysis introduces into the study of its functions and morbid conditions a complexity which clearly necessitates a brief review of various outstanding experimental and other observations if the pathogenesis and treatment of clinical pituitary disorders are to be understood. Experimental investigators of the pituitary have in late years come into agreement to the effect that this organ is essential to life, its complete removal causing death. Removal of the posterior lobe alone causes no symptoms, but removal of a large portion of the anterior lobe is fatal. Removal of a smaller portion of this lobe in dogs has been shown by Crowe, Cushing, and Homans<sup>178</sup> to induce a characteristic state of adiposity coupled with sexual infantilism in young animals and secondary hypoplasia of the generative organs in adults. Polyuria, glycosuria, hypotrichosis, edema of the skin, sub-normal temperature, and mental changes were also commonly noticed. Aschner,<sup>179</sup> in similar experiments in puppies, observed in conjunction with these effects a marked retardation in body growth, reduced general activity, persistence of the puppy type of hair and of the milk teeth, a thick and inelastic skin, failure of the epiphyses to close, a hypoplastic state of the bony skeleton, fatty infiltration of the liver, enlargement of the colloid alveoli in the thyroid gland, unusual thickness of the adrenal cortex, and a persistent thymus. These changes are ascribed mainly to the anterior lobe. Confirmation of the especially prominent influence of this lobe on the essential organs of sex (gonads) has been afforded by the feeding of extracts of the anterior lobe in animals. Goetsch<sup>180</sup> gave such extracts to young rats, and observed premature maturity and functional activity of the ovaries, tubes, and uterine cornua, more marked than follows feeding of corpus luteum. A pair of rats fed anterior pituitary bred earlier and oftener than controls, the effects lasting throughout the adult life of these animals.

The posterior lobe of the pituitary, although experimentally not essential to life as is the anterior lobe, has been shown by Howell<sup>181</sup> to be the chief repository of the blood-pressure-raising principle of this organ. The anterior lobe contains none of this principle, while the pars intermedia, it is said, contains it in less amount than the posterior lobe. Apparently embodied in the colloid secretion of the pars intermedia, the active principle has been supposed by Herring<sup>182</sup> and others to pass through the posterior lobe and thence into the third ventricle of the brain. More recently, however, Blair Bell<sup>183</sup> has asserted that there is not the slightest evidence to the effect that, even if secretion from the neural lobe does pass into the cerebrospinal fluid, this is an essential, beneficial, or even the normal method by which the internal secretion is taken by the animal economy. The blood-stream, on the other hand, is by this investigator considered the real channel for distribution of the pituitary hormone. Again, according to Goetsch,<sup>184</sup> "the cells of the *anterior* lobe without doubt discharge their secretion directly into the large blood and lymph sinuses which are so numerous here and with which the cells are in such intimate contact." According to these conceptions, as the reader will have noticed, the pituitary would have not one, but two secretions.

In contrast with the mental, cutaneous, osseous, dental, thyroid, and other disturbances ascribed to the interference with the *anterior* lobe in experimental hypophysectomy, the disturbances of carbohydrate metabolism attending experimental manipulations of the pituitary have been attributed to involvement of the infundibulum and posterior lobe. Thus, Goetsch, Cushing, and Jacobson<sup>185</sup> found that various operative manipulations of the stalk of the pituitary and of the posterior lobe itself caused a temporary hyperglycemia, with associated diminution of the assimilation limit for ingested carbohydrates and frequently a transient glycosuria. Upon producing a permanent insufficiency of posterior lobe secretion, however, these phenomena were followed by an abnormal and lasting rise in sugar tolerance, which could be removed by intravenous or subcutaneous injection of posterior lobe extract. In the intact animal, indeed, such injections reduce even the normal sugar tolerance, and in sufficient dosage may



cause actual glycosuria. According to Goetsch,<sup>186</sup> the adiposity following hypophysectomy is probably also a result of deficiency of the posterior lobe secretion, rather than due to the interference with the anterior lobe.

As regards excessive posterior lobe secretion, the same investigator concluded from feeding experiments with this lobe that, in contrast to anterior lobe feeding, it has an undoubted retarding influence on the development of the sex glands, as shown in rats by a relatively incomplete growth of the testes after eight and one-half months of posterior lobe feeding. From the facts that the genitals remain normal and young animals continue to develop after ablation of this lobe, Blair Bell<sup>187</sup> is even disposed to assert that the secretion of the posterior lobe is neither beneficial nor essential to life.

Sajous, Sr.,<sup>188</sup> mindful of the prominent nervous components of the posterior lobe, and maintaining the view of a definite, direct connection of this lobe through the infundibulum with overlying nerve tissues, considers the posterior pituitary an important nerve center, capable of influencing the functional activity of all organs through the intermediary of subsidiary centers in the medulla and spinal cord. Being but a co-ordinating center, it is not necessary to life. He also considers it as the sensory organ upon which shocks and traumas in general react most powerfully—a circumstance illustrated in the pathogenesis of acromegaly, fully 20 per cent. of the cases of which arise through some form of accident, physical or mental, frequently a fall upon the head. In regard to the *pars intermedia*, Sajous, Sr., assimilates the nerve-cells found in this portion of the organ by Gentès,<sup>189</sup> and extending by their axons through the posterior lobe to the base of the brain, to the osphradium of sea-dwelling invertebrates—a sensory organ believed by naturalists to represent in ancestral form the pituitary body of vertebrates and to have the function of testing the quality of the water passing over the respiratory organ of these animals. The *pars intermedia*, as the test organ of the blood—the latter a homologue of the sea water in which the tissues of the invertebrates are bathed—is thus conceived of as keeping watch over the purity of the blood, and in the presence of circulating toxic substances as awakening a febrile reaction for their destruction through



nervous excitation of the anterior portion of the floor of the third ventricle, immediately above the pituitary, in which a thermogenic center was found by Ott. This theory attributes the metabolic phenomena witnessed in disorders of the pituitary, such as acromegaly, *adipositas cerebialis*, etc., to the thyroid, adrenals, etc., with which the pituitary is functionally connected by nerve paths, the posterior lobe not being, according to Sajous, Sr., Biedl and others, a secreting organ. Recent observations, too comprehensive to be treated in the present connection, tend increasingly to sustain this view.

The acute pharmacologic effects of extracts of the posterior lobe and *pars intermedia* are so well known as to require little discussion. The chief circulatory action is a direct constriction of the peripheral blood-vessels, causing a pronounced rise in blood-pressure, more prolonged than in the case of adrenalin. Concurrently there is a slowing of the heart-rate, commonly ascribed to a depressor substance in the extract. Other characteristic actions are the now widely utilized oxytocic action; the diuresis, ascribed mainly to renal vasodilatation; the accentuation of peristalsis in the intestine and of the bladder contractions, and the galactagogue effect, variously ascribed to specific stimulation of the secreting mammary cells and to contraction of the smooth muscle around the mammary ducts.

### PITUITARY OVERACTIVITY (HYPER-PITUITARIA).

Definite grouping of pituitary affections into those characterized by overactivity and those with functional insufficiency of the organ is interfered with not only by the fact that an initial overactivity may after a time pass into insufficiency but because the organ is composed of separate lobes, which may be independently involved. Again, overactivity of one lobe may, by enlargement and pressure, cause insufficiency of the other, and enlargement of the organ as a whole may produce "neighborhood symptoms" by pressure on other structures in the vicinity or may induce the general manifestations of increased intracranial pressure. To cases exhibiting a combination of some symptoms of pituitary overactivity (hyperpituitaria or hyperpituitarism) with others of pituitary in-

sufficiency (hypopituitaria), the term dyspituitaria (dyspituitarism) is sometimes applied. Commonly, the symptoms of overactivity or insufficiency of one of the lobes clearly predominate over all other symptoms, thus indicating the part of the organ chiefly affected and the nature of the involvement.

All grades of pituitary overactivity may occur, from a mild, evanescent type to the more pronounced and lasting form resulting from chronic pituitary disease. An increase in weight of the anterior lobe has been shown to occur toward the close of pregnancy, the enlargement of this lobe being due to marked accumulation in it of special, clear, neutrophilic "pregnancy cells," derived from the normal chromophobe cells. The hyperplasia is exceptionally such as to induce a transient bitemporal hemianopsia from pressure on the optic chiasm, together with signs of pituitary overfunction such as thickening of certain parts of the face, enlargement of the hands and feet, and glycosuria, all of which as a rule disappear in a few months after delivery. If, however, involution of the pituitary is incomplete, a species of strumous degeneration of the organ may, after repeated pregnancies, result, eventually inducing symptoms of hypopituitaria.

According to some, syphilis is a frequent cause of pituitary disturbance, presumably at first a hyperpituitaria, later followed by the opposite condition. Harrower<sup>190</sup> believes heredity is an evident factor in such cases, and syphilis in parents and grandparents may leave "an intangible susceptibility" to pituitary disturbance. Either glandular hyperplasia or true tumor formation may be responsible for the more pronounced forms of pituitary overactivity, mainly exemplified in acromegaly.

In classifying conditions of pituitary overactivity according to the lobe chiefly involved, acromegaly and gigantism are generally given as manifestations of overactivity of the anterior lobe. To overactivity of the posterior lobe A. D. Dunn<sup>191</sup> is inclined to ascribe diabetes insipidus. He also recognizes a mixed affection marked by overactivity of the anterior lobe and insufficiency of the posterior, the result being acromegaly coupled with hypophysial obesity. Discussion of acromegaly and gigantism under the heading pituitary overactivity is appropriate because in the initial stages of these affections such

overactivity is believed to occur; the fact is to be remembered, however, that in the later stages of these conditions manifestations of pituitary insufficiency are, on the contrary, likely to exist.

#### **Acromegaly and Gigantism. Tumors of the Pituitary Body.**

Acromegaly or Marie's disease is attributed to pituitary overactivity occurring *after* ossification of the epiphyses. Gigantism or Launois's disease, on the contrary, results where the hyperpituitarism has become established before epiphyseal ossification. Tumors of the pituitary are so merged symptomatically with those of the various stages of acromegaly as to impose the necessity of considering them under the same heading.

In nearly all cases of acromegaly in which examination of the pituitary has been practicable, before or after death, evidences of a hyperplasia or adenomatous process of the organ have been found. Exceptions, as Cushing<sup>192</sup> points out, have been too few to invalidate the pituitary origin of this disease, especially when it is recalled that the osseous changes of acromegaly persist to the end of life even where a pre-existing hyperplasia of the organ has undergone complete involution. As regards the exact nature of the earlier histological changes in acromegaly some degree of uncertainty still prevails, opportunities to examine the organ during the primary stage of overfunction being few. Cushing,<sup>193</sup> among 29 cases of pituitary disease, found 23 instances of epithelial growth or struma originating from the pituitary itself, 20 of these exhibiting large pituitary strumas of chromophobe cells, with coexistent symptoms of secondary *hypopituitarism*. The remaining six cases of pituitary disease showed extrapituitary tumors which had caused pituitary symptoms by compressing this organ. None of these extrapituitary tumors, however, had been associated with actual acromegaly except in one instance; and in the latter a cerebellar cyst and hyperplasia of the anterior lobe were found to coexist. Evidently, therefore, true acromegaly is practically limited to cases in which actual hyperplastic or adenomatous changes in the hypophysis have arisen, and is not likely to occur as a result of pressure on this organ by a tumor arising in other tissues. Cushing points out that the so-called round-celled sarcomas of the pituitary are actually strumas characterized by the presence of large numbers of neutrophilic or

"chromophobe" cells. These cells correspond in particular to the secondary hypopituitary stage of acromegaly, the cells characteristic of *hyperpituitaria* being, on the contrary, con-



Fig. 4.—Mold of the upper extremity in a case of acromegaly. (P. E. Launois.)

sidered to be the acidophile or eosinophile elements of the pituitary.

Of the symptoms of acromegaly and gigantism, some are to be ascribed to the changes in the pituitary itself and the remainder to other influences. Among the manifestations of the primary overfunction of the hypophysis are classed the



skeletal changes, the hypertrophic modifications in the skin and its glandular appendages, and the frequent glycosuria, polyuria, and polydipsia. As to the mentality, Cushing notes in hyperpituitaria certain temperamental changes, in particular irritability, distrust, indecision, coupled with an inability to concentrate and sleeplessness.

In the second stage of hypopituitaria, the bony changes persist practically unmodified, but the thickening of the skin and bogginess of the subcutaneous tissues tend slowly to disappear, and there occur such conditions as marked asthenia, drowsiness, hypothermia, slowing of the pulse-rate, low blood-pressure, abnormal sugar tolerance, and a tendency to obesity. Cushing ascribes all these phenomena to insufficiency of the *posterior* lobe, though admitting that the low blood-pressure, asthenia, and pigmentation noticed in a number of his cases suggest an added secondary inactivity of the adrenals. Sajous, Sr.,<sup>194</sup> had previously emphasized this fact, and considers the manifestations of the second, asthenic stage of acromegaly actually due to hypoadrenia and hypothyroidia, the stimulating impulses to the adrenals and thyroid from the posterior pituitary, which acts as governing center of these organs through nervous connections, being abolished when pressure upon or destruction of the posterior lobe abrogates the functional activity of the latter.

In the primary erethic or sthenic stage of acromegaly stress is likewise laid by Sajous, Sr., on the participation of an induced *hyperadrenia* and *hyperthyroidia*. Support is afforded his view of adrenal participation in the acromegalic syndrome by Cushing's Case XXXII (the giant Turner), whose autopsy, following a period of marked "hypopituitaria" and asthenia, revealed exceedingly diminutive adrenals, with a transverse diameter of only 2 millimeters, and no macroscopical trace of chromaffin elements. In Cushing's Case II, on the other hand, in which death took place from medullary failure after hypophyseal decompression for a recent and progressive *hyperpituitaria*, the adrenals were found large, with their medullas apparently hypertrophic.

The non-glandular manifestations of acromegaly, which occur mainly in the second stage of the disease, include headache, ascribed by Cushing to distention of the capsule of the



pituitary; enlargement and deformation of the sella turcica; visual impairment due to pressure by the growth on the optic chiasm; distortions of the fields of vision and oculomotor disturbances; occasionally anosmia, trigeminal neuralgia, spasticity, uncinat epileptic seizures, frontal lobe manifestations, epistaxis, and an intermittent discharge of mucus into the pharynx. Apart from signs due to direct pressure on neighboring structures by the growth, the customary phenomena of a general increase in intracranial tension may also exist.

In many instances the pituitary disturbance reacts upon the sexual sphere, in particular through hypoplasia or atrophy of the internally secreting tissues of the ovaries or testes. Where



Fig. 5.—Facies in mild acromegaly with associated hyperthyroidia of intermediate severity. (*Moleen.*)

the pituitary disease precedes puberty the secondary sexual characteristics are often but imperfectly acquired, while in pituitary disease occurring in adolescence or later, amenorrhea or impotence and retrogressive change in the essential sex tissues tend to occur.

*Treatment.* Early treatment of acromegaly or gigantism is possible relatively seldom, because the manifestations of the first stage of the disease are, as a rule, not such as will bring the patient to the physician for treatment; or, if headache leads him to seek medical advice, recognition of its true cause may be difficult. Timme,<sup>195</sup> for the early recognition of pituitary disturbance—whether of the hyper or hypo type—emphasizes attention to an “under par” or overstrung condition, headaches, a tendency to drowsiness, hypothermia, epistaxis, deficiency of perspiration even in hot weather, eyes

too close or too far apart to be normal, peculiarities of the hairy growth, and teeth abnormal in character or spacing.

Given a hyperpituitaria recognized early, what corrective non-surgical measures are available?

As yet little has been attempted save the use of the *x*-rays and the administration of palliative drugs for headache. While regarding the *x*-rays merely as an adjunct to operative measures, Cushing<sup>196</sup> refers to observations of Gramegna,<sup>197</sup> Béclère,<sup>198</sup> and Jaugeas,<sup>199</sup> showing that in some pituitary tumors prolonged *x*-ray treatment notably ameliorates the neighborhood symptoms, widening, *e.g.*, the constricted fields of vision. In several of his own cases, in which pressure-symptoms remained marked in spite of partial operative evacuation of the sella turcica, *x*-ray treatment led to an evident diminution of all the pressure manifestations. If such results are obtainable even in the more advanced cases, the method should likewise be of some avail in those detected early. In view of the already well-recognized efficacy of surgical treatment, however, the latter should not be postponed too long where the *x*-ray treatment proves inactive. For the relief of headache in acromegaly antipyrin, acetanilid, and acetyl-salicylic acid have all been used with considerable, though temporary benefit. Where signs of excessive thyroid activity co-exist, the therapeutic measures appropriate in exophthalmic goiter (*q.v.*) may be applied.

The indications for surgical intervention, as defined by Cushing,<sup>200</sup> vary according to the case. To meet general pressure disturbances, which may be so marked as to demand prompt relief before intervention on the pituitary itself can be considered, a subtemporal decompression operation, as for other brain tumors, is recommended. Sixteen of Cushing's 43 operative cases were subjected to this measure, usually before, but occasionally only after an unavailing operation directly on the pituitary. In a single case, an operation to combat functional hyperplasia of the pituitary was undertaken, what was thought to be about one-third of the anterior lobe being removed by Schloffer's transphenoidal route. Subjective discomforts and the thickening and edema of the soft parts were thus greatly improved—though largely returning after a year—but no similar operations were later undertaken, it being

deemed uncertain whether, in the absence of hyperplasia sufficient to cause neighborhood symptoms, such a procedure is worth while from the standpoint of a permanent curative action.

The chief indication for operative treatment, therefore, is for the purpose of relieving the neighborhood symptoms caused by pressure of an hypophyseal tumor on surrounding important structures. Even in this connection the procedure to be carried out must vary in different cases, according to the nature of the lesion. The objects of the operation are either partially to remove the tumor or to provide additional space in the direction of the least important adjoining structures, that the tumor, in its further growth, may be prevented from endangering essential tissues such as the optic nerves.

The earlier methods of surgically approaching the pituitary erred in their complexity, and in the extent to which they exposed the deep structures to infection. According to V. Zachary Cope,<sup>201</sup> two procedures have proven fairly satisfactory, viz., the Hirsch-Cushing submucous nasal method and the fronto-orbital method of Frazier. Cushing gives to A. E. Halstead<sup>202</sup> the credit of first using the sublabial incision in the transphenoidal operation.

In the operation performed by Cushing, after institution of intratracheal anesthesia, the upper lip is raised and a short incision made down to the anterior nasal spine of the superior maxilla, the soft parts scraped back until the cartilaginous septum is exposed, and the septal membrane then separated on each side as in submucous resection. Upon insertion of a retractor 1.8 cm. in breadth and 6 cm. in length, to separate the freed layers of mucous membrane, most of the vomer, the lower edge of the median plate of the ethmoid, and a small strip of the cartilage are removed. A series of dilating plugs, up to a diameter of 1.8 cm., are now introduced to flatten the turbinates slightly, the retractors then withdrawn, and a self-holding, bivalve speculum, with blades about 7 cm. long, inserted. The sphenoidal sinuses having been identified, their anterior and lower walls are chipped away with long-handled nasal rongeurs, the lining mucosa of the sphenoid cells removed, and the floor of the pituitary fossa, forming a protrusion into the cells, also chipped away. With a knife-hook

a crossed incision is finally made in the dura covering the pituitary or growth, and the latter appropriately dealt with. Termination of the operation consists merely in checking bleeding completely, withdrawing the speculum, and closing the lip incision by means of 2 or 3 catgut sutures, without drainage. The two layers of septal mucous membrane, as a rule untorn, fall together, and the entire procedure is thus conducted without actually entering the nasal passages.

As for the mode of dealing with the exposed pituitary lesion, this varies with the type of lesion found. If a mere infrasellar tumor arising from an hypophyseal rest is found, it may be forthwith removed. Usually, however, the pathological tissue is more extensive and situated higher. Cushing found that even in the presence of a large pituitary struma, a simple sellar decompression, as already described, may suffice to relieve pressure against the optic nerves and largely restore vision. In other instances, however, removal of tissue at a later operation is required. Whenever the nature of the tissue exposed is in doubt, especially if there is a possibility that it may be a flattened pituitary, Cushing advises abandoning the operation merely as a sellar decompression, and later approaching the lesion by the intracranial route at a second operation. In the presence of a greatly enlarged sella turcica filled with a large pituitary struma, the part of the tumor occupying the floor of the sella is spooned out—a procedure usually attended with but little bleeding, as such strumas have but little vascularity. These cases occur in considerable numbers. Cases of intrapituitary cyst are less frequent; their treatment consists in evacuation.

Reporting on 95 operative cases, Cushing<sup>203</sup> mentions 37 subtemporal decompressions with 2 fatalities, 8 subtemporal explorations without mortality, 6 subfrontal explorations with 1 death, 16 transphenoidal decompressions with 3 deaths, and 58 transphenoidal extirpations, with 4 deaths. The total operative mortality was thus 8 per cent., and the case mortality, 10.5 per cent. In the last 33 transphenoidal operations there was but 1 death—a mortality of only 3 per cent.

In Hirsch's method the middle turbinates are usually removed as a preliminary measure some days before the main operation. At the latter, performed under local anesthesia,

the initial incision is made through the mucous membrane over the nasal septum, on one or the other side. Special precautions are taken to insure asepsis. The exposure of the pituitary is transphenoidal, as in Cushing's procedure. Of 26 cases thus dealt with, 4 succumbed as a result of the operation.

In spite of Cushing's excellent results, Cope<sup>204</sup> maintains that there must always be a slight danger of meningitis in the submucous pituitary operation, owing to the fact that the sphenoidal ostia open into the nose. After performing three operations by the fronto-orbital method of Frazier, he is inclined to believe the latter more suitable than the submucous procedure in the great majority of cases. In the fronto-orbital method the relation of the frontal sinuses to the supra-orbital margin is first ascertained by transillumination. An osteoplastic flap is then formed in the frontal region, the incision starting at the external angular process, coursing through the eyebrow line to the root of the nose, ascending to within the hair line, turning outward again, and returning to the temporal region on a level with the beginning of the incision. In forming the bone flap the outer portion of the supra-orbital ridge is removed as a wedge-shaped piece. The periosteum is then freed from the roof of the orbit, the roof removed with rongeurs back to the optic foramen, and, if necessary, a small opening made in the dura to permit cerebrospinal fluid to escape, and thus allow greater displacement of the frontal lobe. The orbital contents are drawn downward and outward with flat retractors, the frontal lobe with its dural covering raised, and the dura then incised horizontally, about a centimeter above the base of the skull, sufficiently to admit a retractor and expose the contents of the sella.

Advantages claimed for the fronto-orbital route are, that it provides an aseptic route, that it allows each step of the operation to be performed under direct vision, and that since the primary enlargement of pituitary tumors is towards the brain, the organ is thus an easier object for attack than it is from the infrasellar exposure. Tabulating the operative results of the fronto-orbital method, Cope cites Frazier's series of 4 cases without mortality, Cushing's series of 16 cases with 1 death, and Sargent's 3 cases with 1 death, the total mortality being thus 8.6 per cent.



The possible ultimate results from hypophyseal operations are illustrated in 2 out of 3 cases of pituitary cyst dealt with by Kanavel.<sup>205</sup> Stress is laid on the fact that these cysts actually arise through inclusion of buccal epithelium in the hypophyseal region, the remains of Rathke's pouch, *e.g.*, persisting near the infundibulum, and later proliferating to form cystic or adamantine tumors, hitherto reported under various titles, such as epithelial tumors of the infundibulum, papilloma of the choroid plexus, cystic endothelioma of the pia, adenoma, adenosarcoma, dermoids, etc. The epithelial inclusions forming the starting-point of such tumors reach the pituitary from the craniopharyngeal duct, which in the embryo forms a passage from pharynx to brain cavity traversing the sphenoid bone. In Kanavel's operative procedure the incision is made in the crease of the skin immediately under the nares and alæ of the nose. The nasal spine is then cut, and the mucous membrane carefully raised from the floor of the nose and off of the septum, back to the sphenoid bone, and off from the front of the latter. The pituitary is now exposed through the sphenoid as in Cushing's operation. In the first of Kanavel's cases the cyst found was thoroughly curetted, with the result of bringing the existing typical Fröhlich syndrome to a standstill, and relieving the marked signs of intracranial pressure. Dried pituitary gland was fed for over three years. Six years after the operation the patient was still living and well. The second case succumbed to meningitis after the operation, while the third was operated upon on three successive occasions for pressure relief, with ultimate recovery. Three years after operation there had been no recurrence of symptoms.

Postoperative *x*-ray treatment is often resorted to by Cushing, especially in cases of rapidly enlarging pituitary struma. The exposures are made on alternate days, through the nares and over the temple. A previous sellar decompression permits of direct impingement of the rays on the denuded lesion, while after a temporal decompression the resulting bone defect is believed to render the lateral application of the rays more effectual than would otherwise be the case.

The non-surgical treatment of the secondary, hypopituitaric stage of acromegaly, in particular by organic products, will be described in the succeeding section.

### PITUITARY INSUFFICIENCY (HYPOPITUITARIA).

Apart from the rather numerous cases in which pituitary insufficiency becomes manifest as a secondary phase of hypophysis disease—*i.e.*, in which signs of acromegaly have preceded those of insufficiency—there occur cases in which evidences of deficient pituitary activity characterize the clinical picture from the beginning. Where the morbid process has become established in childhood, the so-called Fröhlich's syndrome, or dystrophia adiposogenitalis, is the result, marked not only by adiposity, but also by a persistence of infantile skeletal and sexual conditions. Where, on the other hand, it begins in the adult, the sexual infantilism is produced through reversion from the conditions normal in this period of life, the adiposity, however, being present as in the Fröhlich syndrome. According to Cushing,<sup>206</sup> true strumas of the hypophysis causing functional insufficiency are most frequently met with in the third and fourth decades of life. Extrapituitary tumors, on the other hand, arising congenitally in the sphenoidal or infundibular regions, are apt to cause symptoms in the earlier decades, compromising the functional activities of the hypophysis from the very beginning.

Characteristic of the Fröhlich disease is the peculiar nature of the adiposity, which, in males, affects a feminine type of distribution. Coupled with it occur hypotrichosis, hypothermia, hypoplasia of the genitals, a low stature, psychoses of varying types, and an abnormally high carbohydrate tolerance. Of these phenomena, the adiposity and the heightened carbohydrate tolerance are particularly ascribed to insufficiency of the posterior lobe by Cushing. The hypothermia, genital modifications, and general undergrowth, on the other hand, are especially attributed to inactivity of the anterior lobe. The adiposity in Fröhlich's syndrome is not infrequently associated with polyphagia, and especially an abnormal desire for sweets. The mental state is characterized by torpor, drowsiness, and constant lassitude. Metabolism is slow, and the urinary solids reduced, though the amount of urine is sometimes increased. The external genitals are of small size, with the pubic hair scanty or wanting. Cryptorchism or an infantile uterus may exist and give rise, respectively, to impotence

or amenorrhea. If amenorrhea be not complete, the menses may appear late, and the flow be irregular or scanty. Women developing hypopituitarism after puberty cease to menstruate. The skin is dry, even in hot weather and during exertion, and generally smooth, though that of the backs of the hands may be wrinkled. The general asthenia affects the involuntary muscles as well as voluntary motion, constipation being a frequent accompaniment. The pulse is often slow and of small volume, the blood-pressure low, and the limbs cold and at times edematous.

Laying stress upon the features permitting of early detection of hypopituitarism, Timme<sup>207</sup> specifies as suggestive of this condition in the preadolescent period a small stature, adiposities, ununited epiphyses, small sexual organs, weak skeletal muscles, malformed and irregularly placed teeth, a prognathous upper maxillary and deficient general bony structure, narrowing of the interval between the eyes, unusually small hands and feet, and weakness of the bladder walls leading to enuresis. The child cries easily, is apt to be cowardly, and gets along with his playmates only with difficulty. He is mentally and physically sluggish, lacks self-confidence, and is backward at school. Where several of these symptoms exist in one individual, Timme believes the presumption strong that pituitary deficiency is present. Occasionally such children have epileptic attacks, at times merely in the form of dreamy periods associated with gustatory and olfactory impressions. In dyspituitarism a general status thymicolymphaticus with excessive adenoid formation, frequent nosebleeds, and intermittent mucous discharges into the pharynx also has been seen.

A special form of preadolescent hypopituitarism—the Lorain type—is distinguished in which, though genital dystrophy and deficient growth exist, adiposity is absent or inconspicuous. According to Cushing's conceptions of the functions of the different portions of the pituitary, this condition would be due chiefly to insufficiency of the anterior lobe. One of his cases illustrating this affection concerned a female patient of 20 years and 6 months, undersized (4 feet 4 inches), weak and delicate, with the bodily proportions of an adult rather than of infancy, but with absence of secondary sexual characteristics. Many similar instances have been reported in which, ap-

parently as a result of decreased functioning of the anterior pituitary early in life, cessation of growth occurred at the age of 10 or 12 years, a species of pituitary (hypophyseal) nanism or infantilism resulting. In the case recorded by Evans and Assinder,<sup>208</sup> the condition was believed to have originated in a fall on the forehead at the age of 5, attacks of headache and drowsiness soon following, growth ceasing at 12 or 14 years. Diabetes insipidus has been noted in association with the pituitary symptoms and ascribed to the disease of this organ.

Special stress has been laid by Sajous, Sr.,<sup>209</sup> on deficient activity of other ductless glands in the pathogenesis of the dystrophia adiposogenitalis, the hypopituitaria entailing a pluriglandular deficiency because of impairment of the function of the pituitary as a governing center of endocrine organs. Thus, the subnormal temperature, low blood-pressure, and occasional skin pigmentation of hypopituitaria are ascribed to secondarily deficient activity of the adrenal medulla; the muscular weakness, scanty hair growth, and undeveloped or infantile genital organs, to deficiency of the adrenal cortex; the adiposis, smoothness, and dryness of the skin to deficiency of the thyroid, and the undersized growth, with deficient skeletal development and imperfect epiphyseal ossification, to pre-adolescent insufficiency of the thymus.

The cause of the pituitary impairment being, as a rule, some form of growth in this organ or in adjacent structures, marked symptoms of pressure on surrounding tissues are to be expected sooner or later. These symptoms have already been enumerated under Pituitary Overactivity, including especially headache, bitemporal hemianopsia, strabismus, the epileptoid seizures already referred to, papilledema, blindness, and sometimes vomiting. Not infrequently, indeed, the pituitary nature of the disease has remained unrecognized until advanced pressure manifestations such as these have been complained of. L. J. Pollock<sup>210</sup> has called attention to the frequency of chronic hydrocephalus as a cause of hypopituitaria, having observed 12 such cases in two years. In all these cases adiposity was the predominating feature. All cases likewise showed delicate, pudgy, tapering hands with broad bases. Genital hypoplasia, however, was observed but once. Each of 6 cases specially examined showed increased carbohydrate tolerance.

## TREATMENT.

The treatment of hypopituitarism comprises (1) the measures necessary to relieve pressure-symptoms caused by the associated pituitary or extrapituitary enlargement or tumor; (2) those required to make up for deficient pituitary functions.

The first group of measures includes both non-operative and operative procedures. Among the former, thyroid preparations have so far played a rather important rôle. While good results from them are by no means regularly to be expected, many cases have been reported in which benefit followed their use, especially as regards the visual impairment. Thus, Thomson and Lang<sup>211</sup> report 4 cases of pituitary disease in which visual failure had occurred some time before thyroid treatment, and yet was largely overcome by this agent. These authors refer also to cases of similar visual improvement witnessed by Fisher and by de Schweinitz, the latter using mercurial inunctions along with large doses of thyroid extract. To account for the benefit from such preparations, Thomson and Lang assert that since in cretins and thyroidectomized animals the pituitary tends to increase in size, one may well believe that a therapeutic hyperthyroidism is sometimes accompanied by a diminution of the size of an enlarged pituitary. Observation seems to have shown, we may add, that this shrinking effect of thyroid treatment on a pituitary enlargement may occur whether the manifestations of pituitary disease at the time be those of hyperpituitarism (acromegaly) or of hypopituitarism. In a case of acromegaly reported by Salmon,<sup>212</sup> all the symptoms became considerably worse when the patient took pituitary extract, yet thyroid medication caused instead a disappearance of the headache, dizziness, and vomiting and an improvement in the mental condition. The view of Sajous, Sr., that it is in reality through other ductless glands, including the thyroid, that the pituitary produces its morbid effects, best explains the beneficial effects of thyroid gland in hypopituitarism.

The x-ray treatment of pituitary enlargement has already been referred to under Hyperpituitarism. Kupferle and von Szily<sup>213</sup> have reported a case of "cancer" of the hypophysis in which, six months after operative removal, vision again began



to fail rapidly and the  $x$ -ray shadow of the recurring tumor increased in size. Even after the beginning of the  $x$ -ray treatment vision diminished further until completely lost. Upon continuing the  $x$ -ray treatment two months, however, sight gradually returned up to 6/24 in one eye, and at the time of writing this condition had been maintained unchanged for seven months. Mesothorium treatment in the pharynx was combined in this case with the  $x$ -rays used externally. T. A. Williams<sup>214</sup> has reported what was apparently a case of neoplasm causing pressure on the hypophysis, with severe central headache, temporary diplopia, dizziness, restricted and interlaced visual fields, increased sugar tolerance, spasticity, and exaggerated reflexes, in which, as a result of numerous  $x$ -ray exposures in the course of eighteen months, headache had ceased for a year, the visual fields expanded over a year and finally returned to normal, the spasticity disappeared, and certain uncinate phenomena experienced by the patient passed off. An apparent difference in the field of applicability for thyroid and  $x$ -ray treatment in such cases is that whereas the former is available only for true pituitary enlargements, the latter is likewise applicable to extrapituitary tumors causing pituitary symptoms merely through pressure on the hypophysis.

In a case of typical hypopituitarism in a boy of 18 recorded by Leszynsky,<sup>215</sup> in which the pressure-symptoms were ascribed to internal hydrocephalus, puncture of the corpus callosum to establish permanent subdural drainage resulted in ten days in a disappearance of all the symptoms of intraventricular pressure, and one year after the treatment a considerable improvement in the visual function had taken place. In a similar case in a child mentioned by Spiller,<sup>216</sup> with marked pituitary symptoms, increased sugar tolerance and eye symptoms, callosal puncture relieved the ocular disturbances, but not the general condition.

By far the most reliable treatment for the pressure-symptoms of pituitary enlargements is surgical operation. The indications and technic of the operative treatment have already been described under Hyperpituitarism (*q.v.*).

For the symptoms of hypopituitarism glandular therapy is manifestly indicated, and its efficacy is supported by animal

experiments, Cushing having found that hypophysectomized dogs could be benefited by injection of pituitary extracts, glandular feeding or hypophyseal implantations. After complete pituitary removal, subcutaneous or intravenous injection of the emulsion of a single fresh pituitary often aroused to apparently normal activity an already hypothermic, somnolent animal. In animals from which nearly all the organ had been removed, organic therapy bridged over the critical post-operative period, giving time for the remaining pituitary tissue to undergo hypertrophy.

In clinical pituitary medication, some degree of differentiation as regards the use of one or the other lobe, or both, is considered possible, the anterior lobe being especially indicated in the presence of insufficient bodily growth, hypothermia, hypotrichosis, and impaired genital activity, while the tendency to adiposity and the abnormal carbohydrate tolerance are most favorably influenced, it is claimed, by ingestion of the posterior lobe. In cases showing signs of deficiency of both the anterior and the posterior lobes, a combined extract of the whole gland is logically indicated. In cases in which the pituitary disturbance has resulted, through primary over-activity, in gigantism or acromegaly, it is of course essential to be certain that the second stage of hypopituitarism has actually been reached before administering pituitary products, lest the treatment bring about aggravation rather than relief. In most cases in which abnormal pressure is being exerted on the pituitary, both lobes of the gland are impaired or destroyed; whole pituitary administration is therefore oftener indicated than the use of but one lobe. In deficient stature due to pituitary insufficiency the chances of improvement from organotherapy vary according to the condition of the epiphyses at the time. If these are as yet ununited, as shown by *x*-ray examination, success in increasing the stature may reasonably be expected, but if they are already completely united, little is to be hoped for from pituitary treatment.

In the successful cases of pituitary feeding, a variety of beneficial results may be noted. The subnormal body temperature tends to return to normal, the blood-pressure to rise, the constipation to be corrected, and the patient's mind to be roused from its lethargy and drowsiness. In one of Cushing's

cases,<sup>217</sup> menstruation became re-established under the treatment after a year of amenorrhea. In a male patient, under surgical and organotherapeutic treatment, libido and potency returned after a long period of abeyance. In some instances, a considerable loss of weight took place, though in but a few was the adiposity completely overcome. In some epileptoid cases, possibly based on pituitary disturbance, improvement occurred under pituitary treatment. Yet in many cases the results of pituitary medication have proved partly disappointing. Kanavel,<sup>218</sup> in an operated case of cyst of the hypophysis with typical Fröhlich syndrome in which pituitary medication was applied for three years, at first with the anterior lobe alone, and later with the whole organ, observed a distinct growth of hair, but no evidence of growth in height (4 feet 9 inches) or size, no appearance of genital function, and no growth in the size of the testicles. The voice did not become more masculine. The excessive adiposity, however, was lost, and the polyuria and acetonuria disappeared, though sugar tolerance remained above normal after two years. The impairment of vision previously existing persisted, but without increasing.

In general, the best results from pituitary medication, which Sajous, Sr., ascribes to the adrenal principle in organic combination indicated by tests to exist in the pituitary, seem obtainable only after prolonged treatment. The dosage, however, is another important consideration, and proper management in this respect was found by Cushing a matter of some difficulty owing to the great apparent variations in the dose requirements in different cases. Whereas a number of cases showed improvement under 18 grains of desiccated bovine pituitary daily, others needed larger amounts, and in 1 case a dosage of 20 grams (300 gr.) daily—prohibitive for continued use owing to its cost—was found necessary to yield subjective benefit. The Armour 0.2-gram (3-gr.) tablets, whether of whole gland or anterior or posterior lobe, actually contain, according to Cushing, but 0.06 gram (1 gr.) of the dried bovine pituitary. A patient using 6 tablets a day would therefore be taking in 0.4 gram (6 gr.) of pituitary substance. Each 0.06 gram (1 gr.) of dried substance, however, represents, according to Cushing, the extractives of about 5 fresh bovine pituitaries.

The patient would, therefore, be ingesting daily the equivalent of 30 bovine pituitaries. To ascertain the proper dosage in a given case, Cushing has resorted to the expedient of giving the subject daily an amount of glucose or levulose sufficient to produce a temporary mellituria in a normal person of equal body weight; meanwhile an increasing amount of pituitary extract—particularly posterior lobe—is given daily, until a trace of sugar appears in the urine. The proper pituitary dosage is thus estimated by the degree of sugar tolerance, that dose of the remedy being adopted which will reduce the patient's excessive tolerance to normal. In some cases, however, even with an enormous dosage of pituitary substance, it proved impossible to produce a levulosuria with normal amounts of levulose.

Because of the prohibitive expense or incomplete effectiveness of pituitary preparations in some cases, polyglandular therapy has been resorted to. Admitting that the malady in hypopituitarism is a polyglandular one, Cushing<sup>219</sup> himself concluded that the administration of extracts of other ductless glands might be of service. Of his patients a number were definitely improved by thyroid treatment. Again, in the case of a eunuchoid giant with asthenia, low blood-pressure, and pigmentation, marked benefit was obtained from adrenal administration. A. S. Cobbledick,<sup>220</sup> in a case of lesion of the left optic tract attributed to a pituitary growth (the feet and hands subsequently enlarging), with distinct evidences of myxedema, noted improvement in the memory and in the patient's numbness when thyroid medication was applied. As already pointed out, Sajous, Sr.,<sup>221</sup> ascribes many of the manifestations of hypopituitarism largely to accompanying secondary deficiencies of the thyroid, adrenals, and thymus. Even for the treatment of an apparently pure hypopituitarism, therefore, he combines with the pituitary medication 0.06 gram (1 gr.) of dried thyroids and 0.12 gram (2 gr.) of dried thymus three times a day. The activity of pituitary substance seems to be enhanced when small doses of thyroid are combined with it.

Subcutaneous or intramuscular administration of pituitary preparations is available where results from oral use prove insufficient. In experimental hypopituitarism in dogs, Goetsch found subcutaneous injections of posterior lobe extract about

four times as effective as introduction by mouth in lowering the assimilation limit for sugars. In one of Cushing's cases<sup>222</sup> subcutaneous injection of 0.2 gram (3 gr.) of boiled anterior lobe extract caused a rise in temperature of nearly 3° F., increased moisture of the skin, a marked lessening of the copious urinary secretion, and a striking resuscitation from the previous stuporous condition, with increased appetite. This patient improved under oral administration. In another case, however,<sup>223</sup> even large doses of pituitary extract by mouth yielded little improvement; yet daily injections of boiled whole gland extract in a dosage representing 0.12 gram (2 gr.) of the dried preparation roused the patient, previously in a state of somnolence approaching unconsciousness, to such an extent that for two weeks he seemed normally active, both mentally and physically. Because of increasing soreness from the injections, these were then discontinued, and the hypophysis of a newborn child, the victim of a birth hemorrhage, was implanted in the subcortex of the temporal lobe at the point of a previous decompression operation. Interruption of the pituitary injections not being followed by a relapse into the former somnolence, the conclusion was reached that the implanted tissue had remained viable.

## DISEASES OF THE PINEAL GLAND.

Located below the splenium of the corpus callosum, with its base directed anteriorly and fixed to the habenular commissure and the posterior commissure above the opening into the aqueduct of Sylvius, the pineal constitutes the remains of a special visual organ in certain invertebrates and low vertebrates. In the higher vertebrates, nearly all the structural peculiarities of a definite sense organ have been lost, and the main histological features are those of a gland. The parenchyma consists of irregular lobes or follicles supported by a small quantity of connective tissue, and the glandular cells themselves contain characteristically large nuclei with their periphery crowded with granules. The so-called "pineal sand," consisting of calcium phosphate and carbonate, occurs merely in the glial stratum overlying the habenular commissure. The pineal, as described by Jordan<sup>224</sup> in sheep fetuses nearly at



full term, is a highly vascular organ, the capillaries in places forming glomerular loops in spaces surrounded by parenchyma more compact than elsewhere in the body. Jordan noted also a few white nerve fibers, and states that the framework of the parenchyma is a reticulum of delicate neuroglia fibers, for the most part continuous with stellate neuroglia cells. Ramon y Cajal<sup>225</sup> also found sympathetic fibers in the gland, forming a plexus close to the gland-cells.

Involutional changes in the pineal body begin relatively early in life. In man it is believed to be functionally most active in the seventh year of life. From then on it undergoes retrograde modifications characterized, according to Jordan's investigations in sheep, by a marked increase in connective tissue and a cellular neuroglia network, and a reduction in the gland parenchyma. The gland does not, however, degenerate completely, and the possibility of functional activity on its part in adult life is not excluded. According to Krabbe, the evidences of involution in extreme old age are no more marked than in the fourteenth year.

Experimental removal of the pineal, a difficult operative procedure, in itself frequently followed by death, has shown, in the successful cases, that the organ is not essential to life. Apart from this, the results have been contradictory. Dandy,<sup>226</sup> removing the pineal in dogs through the third ventricle after incising the splenium of the corpus callosum, noted none of the changes observed by certain other investigators after pinealectomy, and found nothing to sustain the view that the organ has any active endocrine function of importance, either in very young or adult dogs. On the other hand, Horrac,<sup>227</sup> reproducing in guinea-pigs the results previously obtained by a number of other investigators in these and other experimental animals, found that pinealectomized male guinea-pigs show a hastened development of the sexual organs, and females a tendency to breed earlier than controls of the same age and weight. Other experimenters have in some instances noted a temporary acceleration of body growth as a whole after pinealectomy, with subsequent gradual subsidence to the average weight of controls. Experimental results such as these led Pellizzi<sup>228</sup> to the hypothesis that the function of the pineal body is to exercise a moderating action on genito-

somatic development. According to Foa,<sup>229</sup> pinealectomy in rats does not determine an absolute hypertrophy of the testes, but a premature development of them.

Feeding experiments with pineal substance, apparently contradicting the results from pinealectomy, themselves have shown a rapid sexual and somatic development. Dana and Berkeley,<sup>230</sup> administering pineal to kittens and to young rabbits and guinea-pigs, observed a 25 per cent. excess in the weight of these animals over that of controls. McCord,<sup>231</sup> summarizing the results of experiments on 400 young chickens, guinea-pigs, and dogs, reports almost uniformly a more rapid growth of the body than normal, with an early sexual maturity. The excess in rate of growth was most marked—40.9 per cent. excess in eleven weeks in guinea-pigs—in young animals fed with pineal tissue from young animals. No tendency to gigantism, however, was noted, and after maximum size was attained, the pineal substance seemed ineffective. So small an amount of pineal gland as 20 milligrams ( $\frac{1}{3}$  gr.) weekly proved sufficient to stimulate growth beyond the usual rate. The response to pineal in the rate of growth was somewhat more definite in males than in females. The testes from pineal-fed animals were 50 per cent. larger, and showed premature and very active spermatogenesis. Tadpoles fed with pineal tissue at first grew to double the size of controls fed with muscle tissue, and subsequently showed a markedly precocious transformation into adult frogs. Again, paramecia placed in hay infusion with 0.05 per cent. of pineal extract almost invariably showed a more rapid rate of reproduction by transverse splitting than controls. Hoskins,<sup>232</sup> feeding albino rats with thyroid, thymus, hypophysis, and pineal tissues, was led, on the other hand, to conclude that none of these organs has any constant effect upon the growth-rate of young rats. More recently McCord and Allen<sup>233</sup> have reported finding that the pineal contains a substance capable of controlling pigment cell changes in tadpoles. Tadpoles placed in a 1 in 500 pineal emulsion became in five minutes noticeably lighter in color and more translucent than controls; this effect attained its maximum in  $\frac{1}{2}$  an hour and passed off in 3 to 6 hours. Sajous, Sr., does not consider the pineal as a ductless gland, but as a neural organ probably of temporary use in development.

## PINEAL TUMORS.

Krabbe, in a paper published in 1915, asserted that about 70 cases of pineal tumor had been recorded up to that time. The observations of a number of cases of precocious sexual and general body growth in the presence of a pineal tumor led Marburg<sup>234</sup> to recognize a special clinical syndrome associated with pineal dysfunction. As later developed, this syndrome includes: (1) general intracranial manifestations, comprising the various customary evidences of increased intracranial pressure, the latter depending, as a rule, upon a secondary internal hydrocephalus; (2) neighborhood manifestations, comprising especially evidences of encroachment on the corpora quadrigemina and cerebellum, causing oculomotor and pupillary disturbances together with ataxic phenomena; (3) constitutional or metabolic manifestations, ascribed directly to the disturbance of pineal function, and characterized by early growth and maturity of the sexual organs, with pubic and general body hair, and a premature change of voice; an early maturity of thought and speech, due to precocious mental development, and a general overdevelopment of the body such that the appearance of a child of 11 or 12 years may be presented by one actually only 5 or 6 years old. Frankl-Hochwart<sup>235</sup> also was led to describe such a condition.

As a matter of fact, however, only relatively few of the pineal tumor cases have actually exhibited these manifestations of precocity. Only 25 of Krabbe's 70 cases occurred before puberty, and of these only a minority showed such changes. According to some—*e.g.*, Bailey and Jelliffe<sup>236</sup>—the metabolic symptoms that may result from pineal involvement comprise not only sexual precocity, but also adiposis (one form of "*adipositas cerebialis*"), and in some instances cachexia. Again, available facts seem insufficient to demonstrate whether the precocity and other manifestations are actually due to an excessive or deficient function of the pineal. Bailey and Jelliffe state that neither the sexual precocity nor the cachexia can be precisely accounted for, nor can it be stated definitely whether disturbed pineal function alone is capable of causing adiposis, or whether the latter is due to concomitant hydrocephalus of the third ventricle, exerting pressure on

the pituitary. If hypopituitarism is to account for the adiposis, it will not account for the sexual precocity, for in the dys-trophia adiposogenitalis there is a lack of sexual development. Cushing holds that the pituitary functions may easily become modified from pressure exerted on this organ in the presence of a pineal tumor. Bailey and Jelliffe<sup>237</sup> have described a series of manifestations of pineal disease due to such pressure on the various structures surrounding the pineal. According to McCord, furthermore, not enough attention has been paid to possible hormonal relationships of the pineal to other ductless glands. On the whole, the entire subject of pineal pathology must still be considered in an indecisive stage. As McCord<sup>238</sup> has pertinently remarked, experimental evidence is available supporting both the contention that pineal neoplasms retard the activity of this organ and that they increase it.

#### TREATMENT.

In the absence of definite knowledge as to whether the macrogenitosomatic syndrome is due to impaired or to enhanced pineal activity, or even as to whether vagaries of the pineal function are truly responsible at all for such changes, no rational treatment of gross pineal lesions is available. Operative removal of a supposed pineal tumor has so far been considered impracticable in man, owing to the pronounced danger to life (far greater than in pituitary surgery) attending such an operation. Lumbar puncture has at times proven serviceable in relieving pressure-symptoms. Other palliative measures comprise the use of analgesics for the exacerbations of pain, and of various appropriate procedures for tinnitus, cerebellar symptoms, etc. Measures to retard cachexia may be indicated.

#### PINEAL INSUFFICIENCY AND MENTAL RETARDATION.

Dana and Berkeley, with the co-operation of Goddard and Cornell,<sup>239</sup> have reported numerous therapeutic tests (pineal feeding) in both children and the experimental animals, leading to the conclusion that in mental retardation in children, administration of pineal substance is capable of markedly stim-

ulating the sluggish intellectual functions. The preparation of pineal used was made by rubbing up 12 fresh bullock's pineals with sugar, allowing the mixture to dry, and dividing it into 100 capsules. Each capsule corresponded to 150 pounds of live bullock. The dose given was 1 capsule a day. More recent observations apparently have failed to substantiate the earlier therapeutic conception of these investigators. Goddard<sup>240</sup> has announced that later tests among children at the Training School, Vineland, N. J., proved wholly negative.

## ENDOCRINIC DISORDERS OF THE OVARIES.

The endocrinic functions of the ovaries are carried on by the corpus luteum, and the interstitial gland, where present. As is well known, the corpus luteum results from metamorphosis of the collapsed Graafian follicle, after extrusion of the ovum, the latter constituting the *external* secretion of the ovary. The interstitial gland is inconstant in its distribution, not only being absent in certain animal species, but varying in amount in different individuals of the same species, and at different seasons. It consists of epithelioid cells, either disseminated or grouped to form alveoli, in the ovarian stroma. According to Wallert,<sup>241</sup> interstitial cells occur during pregnancy in the human subject.

According to Leo Loeb,<sup>242</sup> a function of the corpus luteum normally is to retard ovulation, for where, at an early period of the sexual cycle, all the corpora lutea are excised, the next ovulation is much accelerated. In pregnancy the life of the corpus luteum is, for some as yet unknown reason, greatly prolonged, and ovulation during pregnancy thereby prevented. This influence of the corpus luteum on ovulation was shown to be exerted in a chemical and not a mechanical way. Another function of the corpus luteum, according to Loeb, is to sensitize the uterine mucosa in order that a maternal placenta be produced upon its excitation, either by the ovum or mechanical means.

In addition to the above "cyclical" functions of the corpus luteum, the internally secreting ovarian tissues exhibit other "non-cyclical" functions, producing a favorable trophic influence on the uterus and mammary glands, both of which under-



go gradual atrophy upon excision of the ovaries and at the menopause. The ovaries also exert a pronounced influence in the development of the secondary sexual characters, the female characters being stimulated through the endocrinic ovarian activity, and the obtrusion of male characters simultaneously prevented. Transplantation of ovaries into castrated males has been found capable of causing enlargement of the mammary glands, and substituting female for male psychic characters.

Whether the internal secretion inducing female characters is produced by the corpus luteum, by interstitial cells, or by both has not as yet been definitely ascertained. The majority of investigators, in experimental administrations of ovarian extracts, found the corpus luteum inactive or less active than other portions of the ovary. A number of observers have noticed that injection of ovarian (or placental) extracts induces hyperemia of the vulva and uterus, together with cellular thickening of the latter. Substances inhibiting the coagulation of blood and inducing hyperemia, thus probably playing a rôle in the production of hemorrhage at menstruation, were found by Schickele<sup>243</sup> both in the ovaries and in the uterine mucous membrane.

That the ovaries must exert some endocrinic influence even during infancy and later childhood is asserted by Frank,<sup>244</sup> on the ground that early castration in animals produces eunuchoid types, characterized by undeveloped sex organs and distinct changes in stature, the bony skeleton, and various ductless glands. On the other hand, at the menopause a physiological cessation of ovarian function takes place, with resulting atrophy of the internal and external genitals, and of the mammary glands, together with characteristic changes in fat and hair distribution as well as of the psychic condition. An artificial menopause through castration in adult life induces similar changes. According to Neumann and Hermann,<sup>245</sup> castration or *x*-ray exposure of the ovaries results in a great increase of the cholesterin or cholesterin compounds in the blood.

That castration directly or indirectly reduces metabolism and oxygen consumption seems to have been shown by the experiments of Loewy and Richter,<sup>246</sup> and by the more recent

work of Marlin and H. Bailey.<sup>247</sup> The latter observed, in combination with a gain in weight after castration in bitches, a lowering of metabolism by from 6 to 17 per cent. An animal with intact thyroid showed a more marked diminution of metabolism than one with the thyroid removed. The experimenters feel that indirect action has a bearing on the reduction of metabolism, and that the presence of a specific stimulus from the ovaries affecting cellular oxidation has not yet been proven. Apart from oxidation, clinical data are available to the effect that the ovaries may exert a control in the metabolism of phosphorus and calcium. Bilateral oöphorectomy apparently causes improvement in osteomalacia by diminishing the excretion of these substances.

The active principle or principles contained in the ovary have not as yet been definitely isolated. Numerous bodies—possibly mixtures of principles—claimed to exert more or less pronounced hormonal effects have been obtained by different investigators through extraction with alcohol and other solvents, filtration, desiccation, etc., but since no two investigators, apparently, have used precisely the same methods, no uniformity of results has been secured. Iscovesco<sup>248</sup> has prepared from the organ a number of what he terms “lipoid” substances, some of which were shown to have the power, upon administration, of stimulating the growth of the ovaries and uterus, while certain others stimulated different organs such as the thyroid, adrenals, heart, kidneys, etc. His chief “hormo-stimulating” lipoid is a yellowish, wax-like substance, which, for experimental and clinical use, was made up in a 2 per cent. solution in oil, and has been commercially available in France. Herrmann<sup>249</sup> obtained from the ovary a similar product, described as a thickly viscous, yellowish oil, giving a decided cholesterin reaction, and becoming brown when exposed to the air, apparently by taking up oxygen. This product was obtained both from the corpus luteum and the placenta. Seitz, Wintz, and Fingerhut<sup>250</sup> found in the corpus luteum one body, the “luteolipoid,” which, when given subcutaneously before and during menstruation, diminishes and abbreviates the flow, and another body, “lipamin,” soluble in water, and which, when injected in animals, augments the development of the reproductive organs, and in women overcomes amenorrhea.

W. H. Morley,<sup>251</sup> after reviewing the work of the above investigators, notes that other workers have often used indiscriminately an "extract" which might be either aqueous, alcoholic, or ethereal, or merely the dried, powdered ovary. In future experimental and clinical work, he considers greater uniformity of preparation, and careful specification of any product used, an urgent necessity. In his own experimental work various extracts were made by exact chemical methods, but yielded only negative results in animals, though one water-soluble extract seemed to be temporarily active clinically. Frank and Rosenbloom<sup>252</sup> obtained from the corpus luteum and placenta extractives soluble in lipid solvents, which, when injected repeatedly into previously castrated female rabbits, caused an enormous hypertrophy and congestion of the uterus, even in animals that had not yet reached the age of puberty. Extracts from the corpus luteum of pregnant animals proved more effective than those from non-pregnant animals. The fact that unfractionated extracts were quantitatively more efficacious than any of the fractions themselves made it seem probable to these investigators—as had already been suggested by others—that the active substance in the ovaries is not a lipid, but is merely carried along by the lipoids.

Lefkowitz and Frank,<sup>253</sup> investigating the ferments of the ovaries, found trypsin, pepsin, lipase, a very small amount of erepsin, and also amylase, the last of these being more abundant in pregnant than non-pregnant animals; the observers do not consider these ferments as bearing on the source or nature of the active substance or substances secreted by the ovary.

That the phenomena of heat in animals are due to secretion of some substance into the blood by the ovaries is confirmed by the experiments of Marshall and Jolly,<sup>254</sup> who observed transitory symptoms of heat in bitches previously not in heat upon injecting blood-serum or transplanting ovaries from bitches in heat.

Clinical studies in ovarian organotherapy have convinced W. P. Graves<sup>255</sup> that preparations of the corpus luteum alone are less efficacious than those of the whole ovary. He lays stress in this connection on the part played by the interstitial cells of the ovary in the elaboration of the ovarian internal secretion. These cells, he deems it probable, correspond to the

lutein cells of the theca interna of the atresic follicles or theca lutein cells of the corpus luteum, and are analogous to the testicular interstitial cells. Ovarian extracts should comprise the stroma, to take advantage of the atresic follicles. Extracts thus made from the ovaries of pregnant animals, with exclusion of the corpora lutea, clinically proved highly efficacious therapeutically in his hands. Extracts from the corpora lutea of pregnancy, on the other hand, proved too toxic for practical use.

Sajous, Sr.,<sup>256</sup> has emphasized the resemblance of the ovarian interstitial cells to the cells of the suprarenal cortex previously pointed out by Mulon, Wallart, and others, and attributes the production of the internal secretion of the ovaries mainly to the dynamism of adrenal rests in the interstitial organ and corpora lutea.

### ENDOCRINIC OVARIAN INSUFFICIENCY.

The effects of insufficiency of the internal secretion of the ovaries vary markedly according to the period of life at which the deficiency arises, as well as various other factors. There is still much discussion as to what symptoms are and what are not due to this form of disturbance.

Where the insufficiency arises during the developmental period, certain characteristic abnormalities of growth may be noted, the extremities, *e.g.*, becoming unusually elongated in relation to the length of the trunk, the sacrum being flattened instead of curved, and the pelvic outlet remaining narrow, instead of expanding, as in the normal female. The distribution of fat and of the hairy covering fails to exhibit in full degree the female characteristics, tending rather toward a eunuchoid or male condition, while the genital organs remain small and infantile. In these or adult cases there also frequently exist nervous and vasomotor symptoms, such as unusual general excitability or nervous sluggishness, flushes, sweats, and dizzy sensations. Altered function of the genital tract shows itself, under such circumstances, in the form of dysmenorrhea with a scanty, irregular flow, or amenorrhea, coupled with sterility. As Frank<sup>257</sup> remarks, secondary hypofunction of the ovaries may occur as a result of thyroid disease, as in myxedema or

exophthalmic goiter; from pituitary disease, as in the second stage of acromegaly and in the dystrophia adiposogenitalis, or in association with insufficiency of the adrenal medulla, as in Addison's disease. Such secondary hypofunction is often preceded by a preliminary period of ovarian hyperactivity.

The well-known symptomatic and other manifestations of the natural menopause constitute, in a sense, the type of the conditions resulting from cessation of the functions of the ovaries at any time after the close of the developmental period. Especially noteworthy are the accumulation of adipose tissue and the vasomotor disturbances. According to Graves,<sup>258</sup> the "ablation symptoms" following extirpation of the uterus and adnexa by improved surgical technic are now much less distressing than formerly, when "disabling and discouraging postoperative complications" were of common occurrence. The only specifically characteristic symptom, according to this observer, is hot flashes, though frequently associated with it, or sometimes occurring independently, are sensations of alternate heat and cold, palpitation, feelings of anxiety, dizziness, and sleeplessness. The majority of patients, states Graves, suffer comparatively slight inconvenience from removal of the ovaries, and many of them none at all—a circumstance which he ascribes to "the subordinate part played by the ovary during maturity in the group of ductless glands." Presenting a table of statistics, this author calls attention to the fact that where, in performing hysterectomy, one or both ovaries are left *in situ*, the subsequent incidence of hot flashes is no less—81 per cent.—than where both ovaries have been removed. This he ascribes to a disturbance of the physiological relationship of the uterus and ovaries, a condition of ovarian "dysfunction" being the result, which often can be corrected only by subsequent removal of the ovaries themselves. Later, however, he states that upon removing ovaries retained at a previous hysterectomy he had found them in every instance "densely adherent, degenerated, and cystic." Such findings, we may note, suggest that the hot flashes he supposes due to ovarian "dysfunction" result merely from destruction of the ovaries, and do not support the view that cessation of ovarian interstitial secretion *per se* is not responsible for the surgical menopause symptoms. Graves also remarks that hot flashes



were reported after removal of the ovaries by many patients who had already passed the menopause. This would tend to show, as he states, that the ovary retains its influence as a secretory organ long after ovulation ceases.

The nervous and mental disturbances following surgical castration in women have been emphasized by Alfred Gordon,<sup>259</sup> who finds that the psychic manifestations may belong to any of the varieties of psychoneuroses, but in their ensemble do not constitute any of the classical forms of psychasthenia. The symptoms generally observed are restlessness; difficulty of self-control; dissatisfaction with all and everything; difficulty of finding contentment in one's own efforts; want of interest in all absorbing subjects and objects; indifference, indolence, and pessimism; sometimes outbreaks of anger, with a tendency to attack. Along with these occurred at times insomnia, functional gastro-intestinal disturbances, headache, vague pains or paresthesias, and occasionally glycosuria and a tendency to obesity. Some of these patients became intolerable to live with, and had to be isolated. A remarkable persistence of the morbid phenomena was also noticed, some patients showing the condition unaltered after ten years.

Many of the nervous phenomena just noted are suggestive of hyperthyroidia, and, as pointed out by Bandler,<sup>260</sup> thyroid disturbances, in particular a marked instability of thyroid function or relative hyperthyroidia, not infrequently accompany the normal climacteric. Among other glandular activities which may be affected, Bandler refers to an excessive functioning of the anterior lobe of the pituitary, ascribed to removal of "inhibition" by the ovary. Overactivity or insufficiency of the posterior pituitary lobe or of the adrenals are also recognized as possible accompaniments, with corresponding metabolic and other symptoms. A severe menopause, according to this author, speaks in general for a poorly balanced endocrine system.

The relationship of the ovaries to disorders of menstruation has been studied, among others, by E. Novak.<sup>261</sup> While believing it a demonstrated fact that the corpus luteum is the cause of normal menstruation, Novak, in careful histological studies of the ovaries from 102 cases, was unable to find any direct relation between the degree of lutein development in

the ovary and the clinical intensity of the menstrual flow. Involvement of the ovary in inflammatory disease has, however, been considered by Hitschmann and Adler<sup>262</sup> a cause of excessive menstruation. The amenorrhea of anemia, phthisis, and other debilitating conditions is possibly due, according to Novak, to an inhibitory effect on the corpus luteum, or more probably, to failure of ovulation itself. This author also regards fibrocystic disease of the ovaries as an important index of ovarian hyperemia and hyperfunction, with excessive menstruation as the most frequent clinical symptom.

Late menstruation, hypoplasia of the uterus and adnexa, and poorly developed secondary sex characteristics may constitute evidence, as Bandler<sup>263</sup> states, of either a primary involvement of the ovaries and genital tract, or of a secondary influence exerted upon them by the thyroid, hypophysis, adrenals, thymus, or other glands. Hypoplastic ovaries, according to this author, may indicate a persistent thymus. The amenorrhea of lactation, ascribed to inhibition of the corpus luteum by a hormone from the actively functioning mammary glands, may, if excessively prolonged, eventuate in an atrophy of the uterus and inhibition of ovarian function which can generally be overcome with ovarian extract, thyroid, iron, and arsenic. Bandler writes also of a decided atrophy of the uterus which occasionally follows a too thorough curettage, and which he ascribes to removal of the stimulating effect of the endometrium, when still *in situ*, on the ovary; this stimulus being lost, the resulting ovarian depression reacts on the uterus. In young women with a more or less mild grade of the adiposogenital syndrome there occurs, along with progressively increasing obesity, a diminution of the ovarian function and of menstruation. An atrophy of the uterus and ovaries takes place which in many cases no method of treatment will overcome. Again, in the second stage of acromegaly, with the accompanying depression of ovarian and uterine function, a pronounced diminution of menstruation is observed.

#### TREATMENT.

There still exist marked differences of opinion as to the actual value of ovarian extracts. This is probably due in part to the variety of methods of preparation, some of the extracts

used having no doubt been actually devoid of physiological activity. Graves, as already mentioned, has pointed out that ovarian extract, for its best effectiveness, should include the interstitial cells, or at least their product. Bucura<sup>264</sup> recommends that in the natural or artificial menopause ovarian extract be given in increasing dosage until all the symptoms complained of disappear, *i.e.*, for a period of one to three years, if necessary. At first he administers the extract for a period of three to eight weeks, next discontinues it for one week, then resumes it, etc., gradually diminishing the period of medication until eventually the remedy is taken but one week in each month. For the relief of the hot flushes or psychoneurotic manifestations of the menopause, or of neurasthenic symptoms during menstrual life, ovarian therapy has found favor in various quarters, though a few continue to deny it any degree of utility. In all cases of diminishing menstruation, says Bandler, ovarian extract is indicated. In late menstruation and an infantile, hypoplastic condition of the generative organs, the initially causative gland should be ascertained and due organic treatment, *e.g.*, thyroid or pituitary preparations given, along with ovarian extract and corpus luteum. As Novak found the corpus luteum mature during that portion of the menstrual cycle in which the endometrium exhibits the premenstrual hypertrophy, special administration of corpus luteum extract is particularly advisable in amenorrhea or oligomenorrhea. Other indications which have been given for corpus luteum or ovarian therapy include sterility not due to pyogenic infection or mechanical obstruction; insufficient compensatory activity of a remaining ovary after its fellow has been removed; repeated abortions not due to disease or mechanical factors; hyperemesis in the early months of pregnancy. Iscovesco<sup>265</sup> used a "lipoid" prepared from the ovary with good results in a number of cases of amenorrhea, dysmenorrhea, disturbances due to ovarian insufficiency or the menopause, chlorosis, and senile debility. A 2 per cent. oily solution of the lipoid was injected deeply into the gluteal muscles in the dose of 1 mil (15 *m.*) daily, or 4 to 6 pills, each containing 0.02 gram ( $\frac{1}{3}$  gr.) of the lipoid, were given by mouth.

Transplantation of ovarian tissue has in many instances been resorted to in order to afford the system a continuous

supply of ovarian product where removal of the ovaries is necessitated by disease or the ovaries have for any other reason become insufficient. According to Graves, who transplanted sections of healthy ovarian tissue in the rectus muscle in cases in which the ovaries had to be removed for pelvic disease, such transplantation has no marked influence on the surgical menopause symptoms. Lydston's work,<sup>266</sup> however, has shown that ovarian implantation is by no means devoid of physiological effects, at least in some cases. In one of his cases, a woman of 59 years, an ovary from a girl of 16, removed twelve hours after death from skull fracture, was implanted eleven hours later into the left labium majus. The patient's hot flushes, sense of exhaustion, somnolence, stiffness of the knees, and long-standing bilateral sciatica disappeared permanently soon after the transplantation. Five months after the procedure the implanted ovary, though diminished in size, could still be distinctly felt. In a second case, in a girl of 17 with dementia præcox, ovarian implantation led to marked improvement in the mental and physical condition. A number of other observers have had experiences with ovarian transplantation which have seemed to indicate physiological value on the part of this procedure. Some, however, have had what they consider disappointing results, and emphasize that a great problem remaining to be solved in this connection is that of overcoming the resistance of the body to grafts from other individuals, whereby such grafts often become absorbed within a relatively short time and fail to gain a foothold in the tissues of the new host.

### ENDOCRINIC OVARIAN OVERACTIVITY.

Menorrhagia and metrorrhagia may at times be considered manifestations of such a state. As Frank<sup>267</sup> points out, the condition, when primary, is, as a rule, limited to the period of sexual maturity, though apt to be most severe toward the beginning and the termination of the period, viz., at the time of puberty and in the preclimacteric. Hyperplasia of the uterine mucosa is a frequent accompaniment, and the uterine muscle itself may be thickened. Some evidence, according to Frank, is at hand indicating that fibroid tumors of the uterus are caused by such a functional hyperplasia. That the second-

ary sex characters may be in some degree modified by unusual activity of the ovarian interstitial tissue is suggested by Riddle's observation that doves can be rendered "overfeminine" in their behavior and characteristics by injections of ovarian material. Bandler<sup>268</sup> recognizes a form of endometrial overgrowth and menorrhagia resulting from ovarian hyperactivity.

Marked precocity of genital development is occasionally met with in the female sex, though more rarely than in male children. Most of these cases are characterized particularly by precocious menstruation, which may occur even before the end of the first year of life. With it may be associated premature mammary enlargement, abnormal size of the external genitalia, a female type of hirsuties, premature change of teeth, and early epiphyseal closure. Excessive development of the body as a whole may also be observed. In Riedel's case, menstruation was observed in a 6-year old child, and the size of the uterus corresponded with that of a normal child of 17. At operation an ovarian sarcoma was found, upon removal of which menstruation ceased. While in this case the ovarian overactivity was primary, such overactivity may in other instances be secondary to hormonal influences from other ductless glands, as in exophthalmic goiter, acromegaly, pineal disease, etc.

#### TREATMENT.

Careful study to ascertain the cause of the excessive ovarian activity, and in particular whether it is primary or secondary, is obviously a necessity. X-ray treatment is probably capable, in suitable dosage, of curbing the condition, though experience thus far has led to the impression that it is more difficult to arrest the function of the ovarian interstitial cells with the rays than to destroy the external secretory function of the ovaries, viz., ovulation. According to Bandler,<sup>269</sup> thymus extract is effectual in the persistent menorrhagia of young girls and in menorrhagia or metrorrhagia following a vaginal operation for uterine prolapse. Where tumors responsible for ovarian hyperactivity exist, either in the ovaries themselves or in other organs, their surgical removal, where practicable, will in some instances be indicated; in others, x-ray treatment may suffice.



## ENDOCRINIC DISORDERS OF THE TESTICLES.

The internal secretion of these organs is now generally held to arise in the interstitial cells of Leydig—strands of polyhedral epithelial cells, of a yellowish color, and frequently containing “crystalloid bodies,” situated in the rather loose connective tissue of the testicles which separates the lobules of the externally secreting or seminiferous tissues. Bouin and Ancel,<sup>270</sup> experimentally ligating the vasa deferentia in animals, found that while the seminiferous tubules atrophied, the interstitial cells remained unaffected, and urged that the activity of these cells was responsible for the development of the secondary sexual characteristics in the male. The same investigators later ascertained that subcutaneous injections of extracts of the interstitial tissue reduced the effects of castration, and that the interstitial cells develop synchronously with the first appearance of spermatogenesis. The conception of the interstitial cells as the source of the secondary sex characteristics was confirmed by Shattock and Seligmann,<sup>271</sup> who observed that occlusion of the vasa deferentia in sheep, with the subsequent atrophy of the seminiferous tissues, does not prevent development of these male characteristics. Foges<sup>272</sup> found that transplanted testes had the same influence in developing secondary sexual characteristics as had normally situated testes, thus showing that spermatogenesis is not a prerequisite to a male type of development.

Experimental work by Allen J. Smith and W. J. Crocker<sup>273</sup> has shown that mere injections of testicular extract are capable at times of developing male secondary sexual characteristics in the opposite sex, even where previous removal of the ovaries has not been performed. Thus, in their experiments, injection of a salt solution extract of cock's testes into hens usually caused an increase in size and brighter coloration of the comb and wattles; greater brilliancy of the neck feathers; in one instance a distinct growth of spurs; diminished egg production; combativeness, and even, in several cases, a tendency to cover other hens after the manner of the cock. According to some, an influence of the testicular extract on trophic nerves may be implicated where actual tissue modifications occur as a

result of its effects. A hormonal action is, however, even more clearly recognized. Launois and Roy<sup>274</sup> state their belief that one of the functions of the generative glands is, in common with certain other ductless glands, to direct, through the agency of the nervous system, the nutrition of certain tissues, especially those of mesodermal source, viz., connective tissue, cartilage, and bone. The effects of castration in delaying the termination of the process of endochondral bone production, abnormal length of the long bones resulting, constitute evidence of an important controlling power of the testes over osseous metabolism.

A distinct stimulating influence of testicular extracts on the neuromuscular mechanism has been proved to exist by Zoth<sup>275</sup> and others. Ergographic tests showed that extract injections materially diminish the muscular and nervous fatigue which follows physical work. Subjective fatigue sensations are likewise reduced.

The most convincing proof of the essential influence of the interstitial testicular tissue in establishing and maintaining the secondary sex characteristics is afforded by the work of Steinach,<sup>276</sup> who, upon transplanting testes in very young animals from their normal position to other situations, saw all the secondary sex characteristics develop at the usual time, with full sexual desire and potency. Upon examination of the transplanted glands, the spermatogenetic structures were found absent, the interstitial tissues, however, being increased in amount. In further experiments, Steinach castrated young male rats and guinea-pigs and transplanted an ovary under the skin or in the peritoneal cavity. In such animals the male characteristics failed to develop, and the male genital organs remained infantile. Female secondary sex characteristics developed to such an extent that the animals were "completely feminized," and were sought by the males as though actually females.

The precise nature of the active substance or substances in testicular extract has not as yet been demonstrated. W. E. Dixon<sup>277</sup> found it to contain a large percentage of nucleoprotein. He states that it also contains a number of extractives, and among these spermine, held by Poehl the chief active constituent. Lecithin and cholesterin were also noted, together with inorganic salts. The significance of the nucleoproteids

in the economy is unknown, though, according to some, they are the source of the beneficial effects obtained from testicular preparations. Spermine, an organic base with the empirical formula  $C_5H_{14}N_2$ , has been held by Poehl and others to have the power to accelerate oxidation of oxidizable bodies, or act as an oxygen carrier. This has led Sajous, Sr., to assimilate spermine to the oxygen-carrying adrenal principle, with which it corresponds in many of its chemical properties. According to Pantchenko, spermine is capable, acting catalytically, of increasing the oxidizing power of the blood and simultaneously activate intraorganic oxidation processes where these are weakened. The fact, however, that spermine has been shown to occur in situations other than the testicles—in fact, according to Dixon, it exists constantly in all body tissues—tends to diminish the significance of spermine as a specific testicular product, and to sustain the views of Sajous, Sr. While present in largest amount in the testes and in nerve-tissue, spermine has been found increased in the blood in leukemia and nervous diseases; it was also discovered early by Leyden in the sputum of asthmatics.

### ENDOCRINIC TESTICULAR INSUFFICIENCY.

Castration in childhood prevents the series of anatomic and functional changes which normally characterize puberty. The skin tends to remain soft and white; the beard does not grow; the larynx fails to enlarge, and the voice remains high-pitched. The muscles do not develop to the extent usual in a vigorous male, while the long bones of the extremities tend to become longer than normal, and the stature of the individual exceeds that which would otherwise have resulted. The reproductive organs as a whole tend to undergo atrophy, but the mammary glands are likely to grow larger than those of the normal male. In general, there is a tendency to abnormal fat deposition, with distribution suggesting the female type. Mentally, these subjects are unusually even-tempered, as compared to other males, but they are distinctly lacking in initiative, are apt to be timorous, submissive, and cowardly, and may be in some degree intellectually blunted. Castration at any time before completion of sexual development regularly causes impotence and loss of sexual desire.

A eunuchoid state alone results, on the other hand, where castration occurs only after completion of sex development, or where the removal of the internally secreting testicular tissue has been only partial. While tendencies as regards fat deposition, deficient hirsuties, smooth skin, and mammary development, similar to those in complete eunuchism, may be noted, the change of voice will generally have already taken place, together with the ossification of the epiphyses, and consequent arrest of the stature at a normal limit. Sexually, where castration occurs after puberty, desire and potency may remain little affected for some time, emissions occurring which resemble normal semen in spite of the absence of spermatozoa therein. This fact is ascribed to the persistence, even after castration, of the various genito-spinal, cerebral, and sympathetic nervous mechanisms related to the reproductive organs, these nervous mechanisms having already become fully developed before the castration. The possibility of a vicarious action of other hormones in such cases has also been given consideration, Lydston, *e.g.*, suggesting that there is a prostatic hormone which functionates vicariously until the prostate itself atrophies as a result of the castration. The penis and other genital organs may also show evidences of atrophy from cessation of the testicular internal secretion.

The causes of such endocrine testicular insufficiency comprise, in particular, traumatic injuries of these organs at any period of life, diseased conditions of the testicles, such as may be due to gonococcal, syphilitic, or tuberculous involvement, or occur as a complication of mumps, and a disturbed nutritive condition of the testicles due primarily to disease of some other ductless gland, as in the dystrophia adiposogenitalis.

#### TREATMENT.

The chief therapeutic measure in interstitial testicular insufficiency consists in supplying the product or products which the organism lacks, either by the use of extracts or, far preferably, by testicular transplantation. As regards spermine, most attention seems to have been attracted to its apparent tonic effect on the circulation and an asserted general improvement of tissue oxidation; little that is definite has been recorded tending to demonstrate direct usefulness of this product in

counteracting interstitial deficiency of the testes. There seems little doubt that a complete extract of these organs is capable of serving much more satisfactorily. While one may safely disregard sweeping dicta which would establish testicular extract as a general panacea for neurasthenia and other forms of systemic debility, little doubt can further prevail that such an extract is frequently capable of limiting, in some degree at least, the prejudicial effects of castration. Where such castration has occurred before puberty, the use of extracts is doubtless totally insufficient to promote development of the missing secondary characteristics. Where the loss of the interstitial testicular secretion has occurred only after puberty, however, testicular organotherapy frequently yields results such as render its use well worth while. Although a manifest influence on the secondary sex characteristics is hardly to be expected, very appreciable results may be obtained in the removal of the circulatory sluggishness, lack of mental alertness, and impairment of certain reflex functions, from which the castrated habitually suffer. According to the experimental demonstrations of Loewy and Richter, the generative glands, whether male or female, exert a distinct influence in stimulating metabolism, or at least, oxidation. The deficient general metabolic activity attending castration is probably the most easily removed by organotherapy of all the effects of cessation of the interstitial secretory function.

Where organic extracts can alone be employed, combination of certain extracts other than testicular with the latter seems to be advantageous. Especially does this apply in the case of extracts of the anterior lobe of the pituitary and of the thyroid gland. Some evidence of the influence of anterior lobe extract on the male reproductive organs is afforded by the observations of Stellwagen.<sup>278</sup> Thyroid substance in moderate dosage seems of advantage in assisting to overcome the sluggish metabolism and adipose tendency which result from castration, whether complete or incomplete. Possibly an extract of the adrenal cortex may likewise prove of some service.

The superiority in the results of testicular transplantation over those of organotherapy appear to be so pronounced that the former is to be strongly advised when practicable. In the clinical field, important pioneer work has been accomplished



in the United States, chiefly by Lydston.<sup>279</sup> Lespinasse had already in 1913 reported a case of impotence relieved by slices of testis 1 millimeter thick transferred from a living subject to another who had been castrated. The implantations were made in the scrotum and rectus muscle. In four days sexual desire and erectile power were restored, and had persisted two years when the patient was last seen. Lydston<sup>280</sup> has reported successful performance of a number of procedures never previously carried out, viz., the first implantation of an entire human testis for therapeutic or experimental purposes; implantation of human sex glands—ovary or testis—taken from the dead body; demonstration of the survival of, and acquirement of new circulation by, implanted human sex glands; implantation of sex glands in dementia præcox, senility, and feminism due to aberrations of testicular structure and function. His experiences appear to him as probably refuting the belief that glands from alien sources, *i.e.*, from other individuals, cannot survive, at least for a considerable period, after implantation. In some cases, indeed, permanent survival of alien glands seemed probable. Glands taken from a living subject, while most desirable, are rarely obtainable, and, according to Lydston, are not more viable than those taken from dead subjects; nothing is lost by ordinary refrigeration for twenty-four to forty-eight hours before implantation. Portions of glands, when implanted, are to a certain degree serviceable, according to conditions and dose. The benefits of implantation probably accrue irrespective of the site of implantation. The procedure was found to have a very useful field in the treatment of impotence, and it is believed implantation, with or without anastomosis with the vas deferens, may have a certain range of usefulness in sterility. Sexual inversions or perversions, certain cases of cryptorchidism and imperfect testicular development, or atrophy from disease, are deemed a promising field for the procedure. Stress is also laid on the general physiologic efficiency, and hence individual and social efficiency, accruing from testicular implantation. At least one of Lydston's cases plainly shows a favorable influence of testicular implantation in leading to the establishment of male secondary sex characteristics in spite of complete destruction of the testicles before puberty.

In a case of grafting reported by R. T. Morris,<sup>281</sup> 3 wedge-shaped segments of testicle, each about 3 mm. in thickness, were implanted, respectively, beneath the sheaths of the left and right rectus abdominis muscles and into the scrotum of a man aged 27 who had lost both organs as a result of orchitis complicating mumps at the age of 13. Although the tissue implanted in the scrotum gradually diminished in size, the remaining vestige of atrophied testicle, along with the epididymis and spermatic cord, showed a synchronous considerable enlargement. Distinct evidences of stimulation of the sexual function were observed, but the patient's youthful voice had not yet undergone any change one year after the initial implantation, though a second graft had been introduced about ten months after the first. Lichtenstern<sup>282</sup> has reported a case of gun-shot wound necessitating removal of the testicles in a man of 28 years. Two weeks later sexual desire disappeared. An increase of the fatty tissue about the neck was noted, together with thinning of the beard. A testicle from a case of inguinal hernia was divided into two parts and sutured in the abdominal muscles on opposite sides. Libido and erectile power reappeared on the sixth day, the beard began to grow thicker again, and the tissues of the neck returned to normal.

Lydston's experiences have shown that whereas a relatively large dose of the testicular product is requisite for the development of the secondary sex characteristics, a very small dosage is sufficient to preserve virility. Once virility has been established, an extremely small dose will preserve the psychosexual and physiosexual characteristics essential to potency. Stress is laid by Lydston on the preservation of a small quantity of normal testicular tissue where removal of the greater portion of these organs becomes necessary because of local disease.

### ENDOCRINIC TESTICULAR OVERACTIVITY.

Little is known concerning this condition, which, however, at times probably exists. The sexual precocity at times associated with hypernephromatous tumors or growths of the pineal gland is presumably related to excessive activity of the interstitial testicular tissue. It seems a question whether among normal males, excessive combativeness and a tendency to out-

bursts of violent temper may not at times be actually a manifestation of unusual activity of the interstitial gland cells, constituting, as it were, an exaggerated form of a normal secondary sex characteristic. This, according to Sajous, Sr., is apt to attend the excessive use of meat. Unusual hirsuties may possibly be a similar manifestation. Disturbances of other ductless glands will, however, also require consideration in such cases. Satyriasis seems to be due in most cases, if not always, to causes other than endocrinic testicular overactivity.

### TREATMENT.

This will necessarily vary according to the nature of the case, and in most instances will be merely palliative. The bromids are usually helpful, in addition to marked reduction of meat in the diet. The  $x$ -rays might prove of service to reduce the interstitial testicular function, where palliatives fail. In some instances tumor formation in an organ other than the testes might indicate surgical removal of the tumor, as the primary cause of the exaggerated sexual manifestations.

### BIBLIOGRAPHY.

1. Stoerk and von Haberer: *Arch. f. mikrosk. Anat.*, 1908, lxxii, 481.
2. Srdinko: *Sitzungsab. d. böhm. Kaiser-Joseph-Akad.*, Prag, 1905, 16, 28.
3. Sajous: *Internal Secretions and the Principles of Medicine*, Ed. 7, Philadelphia, 1916, i, 58.
4. Bernstein and Falta: *Lancet*, 1912, clxxxii, 1787.
5. Abelous and Langlois: *Lancet*, 1898, clv, 494.
6. Jackson, D. E.: *Jour. of Pharmacol. and Exper. Therap.*, 1912, iv, 59.
7. Stewart and Rogoff: *Jour. of Pharmacol. and Exper. Therap.*, 1916, xxiv, 479.
8. Hoskins and McPeck: *Jour. Amer. Med. Assn.*, 1913, ix, 1777.
9. Mackenzie: *Arch. of Internal Med.*, 1917, xix, 593.
10. Sajous: *Internal Secretions*, Philadelphia, 1916, i, 70.
11. Gaskell: *Phil. Trans. Royal Soc.*, London, 1914, ccv, 153.
12. Sajous: *Internal Secretions*, 1903, i.
13. Gley: *C. r. de l'Acad. des sci.*, 1914, clviii, 2008.
14. Cannon and Cattell: *Amer. Jour. of Physiol.*, 1916, xli, 74.
15. Voegtlin and Macht: *Jour. Amer. Med. Assn.*, 1913, lxi, 2136.
16. Iscovesco: *Bull et mém. de la Soc. méd. des Hôp. de Paris*, 1912, xxviii, 37.

17. de Mira: C. r. de la Soc. de biol., 1912, lxxiii, 377.
18. Abelous and Langlois: *Loc. cit.*
19. Merklen: Bull. et mém. de la Soc. méd. des hôp. de Paris, 1917, xxxiii, 1808.
20. Carl: Berl. klin. Woch., 1911, xxxvii, 1827.
21. Sézary: Paris méd., 1912, ii, 512.
22. Mann: Jour. Exper. Med, 1916, xxiii, 203.
23. Landau: St. Petersburg, med. Woch., 1908, xxxiii, 249.
24. D'Elsnitz: Presse méd., 1913, xxi, 755.
25. Morat and Doyon: Traité de Physiologie, 1904, 441.
26. Josué: Paris med., 1917, vii, 1.
27. Williams: Arch. of Diag., 1915, viii, 355.
28. Hopton: Brit. Med. Jour., 1914, ii, 669.
29. Marshall and Davis: Jour. of Pharmacol. and Exper. Therap., 1916, viii, 525.
30. Comessatti: Rivista crit. di clin. med., 1910, xi, 49.
31. Tschoboksaroff: Berl. klin. Woch., 1911, xlviii, 1027.
32. Moltschanow: Jahrb. f. Kinderheilk., 1912, lxxvi, 200.
33. Beresnegowski: Beitr. z. klin. Chir., 1914, xci, 403.
34. Remlinger and Dumas: Compt. rend. Soc. de biol., 1915, lxxviii, 438.
35. Sergent: Bull. de l'Acad. de méd., 1915, 36, 268.
36. Moizard: J. de méd. et chir. prat., 1909, lxxx, 881.
37. Kirchheim: Münch. med. Woch., 1910, lvii, 2694.
38. Mansvetova: Roussky Vrach, 1914, xiii, 874.
39. Sergent: *Loc. cit.*
40. Josué: *Loc. cit.*
41. Rohmer: Münch. med. Woch., 1914, lxi, 1336.
42. Mackenzie: Arch. of Internal Med., 1917, xix, 4.
43. Lippmann: Med. Klinik, 1913, ix, 935.
44. Busch, Leonard and Wright: Jour. Amer. Med. Assn., 1908, li, 640.
45. Courmont: Congr. de méd. interne, Montpellier, 1898.
46. Morton: Austral. Med. Jour., 1912, ii; n. s., 573.
47. Sergent: Jour. de méd. et de chir. prat., 1913, lxxxiv, 410.
48. Adams: Pract., 1903, lxxii, 472.
49. Bate: Amer. Pract. and News, 1899, xxviii, 90.
50. Suckling: Brit. Med. Jour., 1898, i, 1388.
51. Daland: Penna. Med. Jour., 1917, xxi, 183.
52. Pitres and Gautrelet: C. r. de la Soc. de biol., 1910, lxxviii, 1092.
53. Gaucher and Gougerot: Ann. des mal. vén., 1911, vi, 321.
54. Munro: Brit. Med. Jour., 1912, i, 665.
55. Lowry: Med. Klinik, 1914, x, 1800.
56. Bernard and Bigart: Jour. de physiôl. et de pathol. gén., 1902, iv, 1014.
57. Brown and Pearce: Jour. of Exper. Med., 1915, xxii, No. 5.
58. Loeper and Oppenheim: in Malad. des Reins et des Caps. Sur., by Debove, etc., 1906, 738.
59. Dudgeon: Amer. Jour. Med. Sci., 1904, cxxvii, 134.
60. Küttner: Münch. med. Woch., 1912, lix, 2422.

61. Glynn and Hewetson: Jour. of Path. and Bact., 1913, xviii, 81.
62. Israel: Deut. med. Woch., 1905, xxxi, 1745.
63. Dunn: Jour. of Path. and Bact., 1915, xix, 456.
64. Michon: Bull. et mém. Soc. de chir. de Paris, 1913, xxxix, 1764.
65. Tuffier: Bull. de l'Acad. de méd., 1914, lxxi, 726.
66. Auvray: Rev. de gyn. et de chir. abdom., 1912, xviii, 353.
67. Bourcy and Legueu: Jour. d'urol., 1912, i, 181.
68. Nicaise: Bull. méd., 1911, xxv, 987.
69. Bensley: Amer. Jour. of Anat., 1916, xix, 37.
70. Kendall: Jour. Amer. Med. Assn., 1916, lxvi, 811.
71. Marine: Jour. Biol. Chem., 1915, xxii, 547.
72. Rogoff: Jour. of Pharmacol. and Exp. Therap., 1916, ix, 1.
73. Cannon and Cattell: Boston Med. and Surg. Jour., 1916, clxxiv, 138.
74. Sajous: Internal Secretions, Ed. 1, 1903, i.
75. Kendall: *Loc. cit.*
76. Sajous: *Loc. cit.*
77. Chittenden: Trans. Congr. Amer. Phys. and Surgs., 1897, iv, 93.
78. Youchtchenko: Arch. des sci. biol. de l'inst. imp. de méd. exp. à St. Petersbourg, 1910, xv, 173.
79. Albertoni and Tizzoni: Cited by Maragliano, Gaz. degli osped., 1894, xv, 1328.
80. Masoin: Bull. de l'Acad. de méd. de Belgique, 1895, i, 88.
81. Burge, Kennedy and Neill: Amer. Jour. of Physiol., 1917, xliii, 433.
82. Thiele and Nehring: Zeit. f. klin. Med., 1896, xxx, 41.
83. Kendall: *Loc. cit.*
84. Sajous: Internal Secretions, Ed. 1, i, 762.
85. Fassin: C. r. de la Soc. de biol., 1907, lxii, 388, 467, and 647.
86. Léopold-Lévi and de Rothschild: Physio-pathology of the Thyroid Body, etc., 1911.
87. Hunt: Jour. Amer. Med. Assn., 1907, xlix, 240.
88. Jeandelize and Perrin: Réunion biol. de Nancy; cited by Parhon and Golstein: Sécrétions Internes, Paris, 1899.
89. Palmer: Amer. Jour. of Physiol., 1917, xlii, 572.
90. Salzman: Amer. Jour. of Obstet., 1916, lxxiv, 812.
91. Blanc: Progrès méd., 1917, xxxii, 95.
92. Petzetakis: Presse méd., 1917, xxv, 12.
93. Cristiani: Sem. méd., 1904, xxiv, 81.
94. Charrin and Cristiani: Bull. méd., 1906, xx, 630.
95. Cristiani: Bull. de l'Acad. de méd., 1907, lviii, 7.
96. Kummer: Rev. méd. de la Suisse rom., 1916, xxxvi, 241.
97. Goodman: Amer. Jour. Med. Sci., 1916, clii, 348.
98. Kocher: Deut. med. Woch., 1914, xl, 998.
99. Theisen: Ann. of Otol., Rhinol., and Laryngol., 1914, xxiii, 1.
100. Kocher: Keen's Surgery, 1908, iii, 380.
101. DeMassary: Paris méd., 1914, iv, 203.
102. Sajous: Internal Secretions, 1903, i, 164.
103. Chittenden: *Loc. cit.*
104. DuBois: Arch. of Internal Med., 1916, xvii, 915.



105. Cannon: Amer. Jour. Physiol., 1916, xli, 58.
106. Pietrowicz: Jour. Amer. Med. Assn., 1916, lxvi, 141.
107. Hallion and Carrion: Bull. gén. de therap., 1907, 459.
108. Pal: Rev. de therap., Feb. 28, 1914, lxxxi, 152.
109. Richter: Med. Herald, 1916, xxxv, 307.
110. Ochsner: Annals of Surg., 1916, lxiv, 385.
111. Solis-Cohen: Amer. Jour. Med. Sci., 1912, cxliv, 13.
112. Porter: Jour. Amer. Med. Assn., 1913, lxi, 93.
113. Watson: Jour. Amer. Med. Assn., 1914, lxii, 126; 1915, lxv, 1102.
114. Simpson: So. Med. Jour., 1916, ix, 857.
115. Fischer: Ugeskr. f. Laegev., 1916, lxxviii, 1755.
116. Pfahler and Zulick: Amer. Jour. Röntgenol., 1916, iii, 63.
117. Berkman: St. Paul Med. Jour., 1916, xviii, 300.
118. Judd and Pemberton: Med. Press and Circular, 1916, cii, 125.
119. McCarrison: Lancet, 1912, clxxxii, 357.
120. Langmead: Lancet, 1913, clxxxiv, 1312.
121. Haslebach: Corresp. f. schweizer Aerzte, 1917, xlvii, 243.
122. Watson: Texas Med. Jour., 1916, xxxii, 153.
123. Mayo: Jour. Amer. Med. Assn., 1913, lxi, 10.
124. Pool: Surg., Gynec., and Obstet., 1917, xxiv, 150.
125. McCarrison: Indian Jour. Med. Research, 1916, iv, 183.
126. Beebe: Jour. Amer. Med. Assn., 1911, lvi, 658.
127. Vassale and Generali: Riv. di patol. nerv. e ment., 1896, i, 95, 249.
128. Gley: Brit. Med. Jour., 1901, ii, 771.
129. Tanberg: Jour. of Exper. Med., 1916, xxiv, 547.
130. Sajous: Internal Secretions, Ed. 7, i, 150-168.
131. Jeandelize: Insuffisance thyroïdienne et parathyroïdienne, 1903, 45.
132. MacCallum and Voegtlin: Johns Hopkins Hosp. Bull., 1908, xix, 91.
133. Kendall: Jour. Amer. Med. Assn., 1916, lxvi, 811.
134. MacCallum and Voegtlin: *Loc. cit.*
135. Wilson, Stearns, etc.: Jour. Biol. Chem., 1915, xxiii, 89, 123.
136. Voegtlin: Surg., Gynec., and Obstet., 1917, xxv, 244.
137. Kendall: *Loc. cit.*
138. Koch: Jour. of Lab. and Clin. Med., 1916, i, 299.
139. Winternitz: Johns Hopkins Hosp. Bull., 1909, xx, 269.
140. Brantham: Amer. Jour. Med. Sci., 1908, xlviii, 161.
141. Härbitz: Jour. Med. Research, 1915, xxxii, 361.
142. Berkeley: Med. Rec., 1910, lxxviii, 1146.
143. Hammar: Anat. Anz., 1905, xxvii, 23, 41.
144. Biedl: Internal Secretary Organs, 1913, 112.
145. Pappenheimer: Surg., Gynec., and Obstet., 1917, xxv, 276.
146. Huiskamp: Zeit. f. physiol. Chemie, 1901, xxxii, 145.
147. Nordmann: Arch. f. Chir., 1914, cxx, 172.
148. Howland, McClure and Park: Trans. Amer. Ped. Soc., 1914, xxvi, 274.
149. Pappenheimer: *Loc. cit.*
150. Morel: Paris méd., 1914, iv, 161.
151. Gudernatsch: Zentralbl. f. Physiol., 1912, xxvi, 323.

152. Wilcox: Boston Med. and Surg. Jour., 1908, clix, 210.
153. Sajous: Internal Secretions, Ed. 7, 1916, 288.
154. Pappenheimer: *Loc. cit.*
155. Hart and Nordmann: Berl. klin. Woch., 1910, xlv, 814.
156. Sajous: Internal Secretions, Ed. 7, 1916, i, 289.
157. Leeper: Rev. of Neurol. and Psychol., 1912, x, 11.
158. Sajous: Internal Secretions, Ed. 7, 1916, i, 293.
159. Dock: Ohio State Med. Jour., 1915, xi, 555.
160. Dock: *Loc. cit.*
161. Friedlander: Amer. Jour. Dis. of Children, 1917, xiv, 40.
162. Falls: Surg., Gynec. and Obstet., 1916, xxii, 712.
163. Hammar: Svenska Läk. Handling., Sept., 1916.
164. Nordmann: Arch. f. klin. Chir., 1914, cvi, 172.
165. Grossman: N. Y. Med. Jour., 1915, cii, 1089.
166. Schoeppler: Centralbl. f. allgem. Path. u. path. Anat., 1914, xxv, 289.
167. Falls: *Loc. cit.*
168. Garrod: Brit. Med. Jour., 1914, ii, 571.
169. Griffith: Jour. Amer. Med. Assn., 1913, lx, 1023.
170. Gismondi: Gazz. d. Osped. e d. Clin., 1916, xxxvii, 1634.
171. Gismondi: *Loc. cit.*
172. Friedlander: *Loc. cit.*
173. Cook: Boston Med. and Surg. Jour., 1916, clxxv, 483.
174. Gismondi: *Loc. cit.*
175. Parker: Amer. Jour. Dis. of Children, 1913, v, 2.
176. Falls: *Loc. cit.*
177. Berkley: Brain, 1894, xvii, 515.
178. Crowe, Cushing and Homans: Bull. Johns Hopkins Hosp., 1910, xxi, 160.
179. Aschner: Arch. f. d. ges. Physiol., 1912, cxlvi, 1.
180. Goetsch: Bull. Johns Hopkins Hosp., 1916, xxvii, 29.
181. Howell: Jour. Exper. Med., 1898, iii, 245.
182. Herring: Quart. Jour. Exper. Physiol., 1908, i, 121.
183. Blair Bell: Quart. Jour. Exper. Physiol., 1917, xi, 77.
184. Goetsch: Surg., Gynec. and Obstet., 1917, xxv, 229.
185. Goetsch, Cushing and Jacobson: Bull. Johns Hopkins Hosp., 1911, xxii, 165.
186. Goetsch: *Loc. cit.*
187. Blair Bell: *Loc. cit.*
188. Sajous: Internal Secretions, Ed. 7, 598.
189. Gentès: C. r. de la Soc. de biol., 1903, lv, 100.
190. Harrower: Med. Herald, 1916, xxv, 361.
191. Dunn: Amer. Jour. Med. Sci., 1914, cxlviii, 114.
192. Cushing: The Pituitary Body and its Disorders, Phila., 1912, 28.
193. Cushing: *Op. cit.*, 291.
194. Sajous: Internal Secretions, Ed. 7, 1916, 609.
195. Timme: N. Y. Med. Jour., 1915, cii, 801.
196. Cushing: *Op. cit.*, p. 321.

197. Gramegna: Rev. neurol., 1909, xvii, 15.
198. Bécclère: Arch. Röntgen Ray, 1909-10, xiv, 142.
199. Jaugeas: Les rayons de Röntgen dans le diagnostic et traitement des tumeurs hypophysaires, etc., Paris, 1909.
200. Cushing: *Op. cit.*, p. 291.
201. Cope: Lancet, 1916, cxc, 601.
202. Halstead: Trans. Amer. Surg. Assn., 1910, xxviii, 73.
203. Cushing: Jour. Amer. Med. Assn., 1914, lxiii, 1515.
204. Cope: *Loc. cit.*
205. Kanavel: Surg., Gynec. and Obstet., 1918, xxvi, 61.
206. Cushing: *Op. cit.*, p. 42.
207. Timme: *Loc. cit.*
208. Evans and Assinder: Birmingham Med. Rev., 1916, lxxx, 1.
209. Sajous: Internal Secretions, Ed. 7, 1916, i, 308.
210. Pollock: Jour. Amer. Med. Assn., 1915, lxiv, 395.
211. Thomson and Lang: Med. Chron., 1914, lviii, 321.
212. Salomon: Presse méd., 1913, xxi, 1019.
213. Küpferle and von Szily: Deut. med. Woch., 1915, xli, 911.
214. Williams: Wash. Med. Annals, 1916, xv, 103.
215. Leszynsky: Med. Rec., 1915, lxxxviii, 172.
216. Spiller: *Loc. cit.*
217. Cushing: *Op. cit.*, p. 318.
218. Kanavel: *Loc. cit.*
219. Cushing: *Op. cit.*, p. 316.
220. Cobbledick: Lancet, 1916, cxc, 1262.
221. Sajous: *Op. cit.*, 1916, i, 308.
222. Cushing: *Op. cit.*, p. 47.
223. Cushing: *Op. cit.*, p. 320.
224. Jordan: Amer. Jour. Anat., 1911, xii, 249.
225. Ramon y Cajal: Soc. Espan. Hist. Nat., 1895.
226. Dandy: Jour. Exper. Med., 1915, xxii, 2.
227. Horrax: Arch. of Internal Med., 1916, xvii, 607.
228. Pellizzi: Riv. ital. d. neuropatol., 1910, iii, 193.
229. Foa: Arch. ital. de biol., 1914, lxi, 79.
230. Dana and Berkeley: Med. Rec., 1913, lxxxiii, 835.
231. McCord: Surg., Gynec. and Obstet., 1917, xxv, 250.
232. Hoskins: Jour. Exper. Zool., 1916, xxi, 295.
233. McCord and Allen: Jour. Exper. Zool., 1917, xxiii, 207.
234. Marburg: Wiener med. Woch., 1907, lxii, 2512.
235. Frankl-Hochwart: Wiener med. Woch., 1910, lx, 505.
236. Bailey and Jelliffe: Arch. of Internal Med., 1911, viii, 851.
237. Bailey and Jelliffe: *Loc. cit.*
238. McCord: Interstate Med. Jour., 1915, xxii, 354.
239. Goddard and Cornell: Med. Rec., 1913, lxxxiii, 835.
240. Goddard: Jour. Amer. Med. Assn., 1917, lxviii, 1340.
241. Wallert: Zeit. f. Geburts. u. Gynäk., 1906, lxiii, 520.
242. Loeb: Surg., Gynec. and Obstet., 1917, xxv, 300.
243. Schickele: Arch. f. Gynäk., 1912, lxxxvii, 409.

244. Frank: Surg., Gynec. and Obstet., 1917, xxv, 225.
245. Neumann and Hermann: Wiener klin. Woch., 1911, xxiv, 411.
246. Loewy and Richter: Arch. f. Anat. u. Physiol., 1899, 174.
247. Murlin and Bailey: Surg., Gynec. and Obstet., 1917, xxv, 332.
248. Iscovesco: Compt. rend. de la Soc. de biol., Paris, 1912, lxxii, 858.
249. Herrmann: Monatsch. f. Geburtsh. u. Gynäk., 1915, xli, 1.
250. Seitz, Wintz and Fingerhut: Münch. med. Woch., 1914, lxi, 1657 and 1734.
251. Morley: Surg., Gynec. and Obstet., 1917, xxv, 324.
252. Frank and Rosenbloom: Surg., Gynec. and Obstet., 1915, xxi, 646.
253. Lefkowitz and Frank: Surg., Gynec. and Obstet., 1911, xiii, 36.
254. Marshall and Jolly: Edinb. Med. Jour., 1907, xxi, 219.
255. Graves: N. Y. Med. Jour., 1917, cv, 1262.
256. Sajous: Internal Secretions, Ed. 7, 1916, i, 481.
257. Frank: *Loc. cit.*
258. Graves: Surg., Gynec. and Obstet., 1917, xxv, 315.
259. Gordon: Jour. Amer. Med. Assn., 1914, lxiii, 1345.
260. Bandler: Amer. Jour. of Obstet., 1917, lxxvi, 644.
261. Novak: Jour. Amer. Med. Assn., 1916, lxvii, 1285.
262. Hitschmann and Adler: Arch. f. Gynäk., 1913, c, 233.
263. Bandler: *Loc. cit.*
264. Bucura: Zentralbl. f. Gynäk., 1916, xl, 816.
265. Iscovesco: Presse méd., 1912, xx, 845.
266. Lydston: Impotence and Sterility, with Aberrations of the Sexual Function and Sex-gland Implantation; Chicago, 1917.
267. Frank: *Loc. cit.*
268. Bandler: *Loc. cit.*
269. Bandler: *Loc. cit.*
270. Bouin and Ancel: Compt. rend. Soc. de biol., 1903, lv, 1397.
271. Shattock and Seligmann: Brit. Med. Jour., 1904, ii, Suppl., 11.
272. Foges: Pflügers Arch., 1903, xciii, 39.
273. Smith and Crocker: N. Y. Med. Jour., 1913, xcvi, 1.
274. Launois and Roy: Etudes biologiques sur les géants, Paris, 1904.
275. Zoth: Arch. f. d. ges. Physiol., 1896, lxii, 335.
276. Steinach: Arch. f. d. ges. Physiol., 1912, cxliv, 71.
277. Dixon: Pract., 1901, lxvi, 517.
278. Stellwagen: N. Y. Med. Jour., 1916, civ, 879.
279. Lydston: *Op. cit.*
280. Lydston: *Op. cit.*
281. Morris: Jour. Amer. Med. Assn., 1916, lxvii, 741.
282. Lichtenstern: Med. Klin., 1916, xii, 27.





# Diseases of the Cardiovascular System

BY

J. NORMAN HENRY, M.D.,

Visiting Physician, Pennsylvania Hospital; Formerly Clinical Professor  
of Medicine, Woman's Medical College of Pennsylvania; formerly  
Assistant Physician, Philadelphia Hospital;

AND

S. CALVIN SMITH, Sc.M., M.D.,

Instructor in Medicine, Jefferson Medical College; Acting Chief Clin-  
ical Assistant, Medical Clinic, Jefferson Hospital, Philadelphia.



# Diseases of the Cardiovascular System.

---

## FOREWORD.

THE time has come when diseases of the heart are to be rewritten in the new nomenclature and broader understanding which has been brought to us by those modern instruments of clinical precision, the polygraph and the electrocardiograph. This work, coming as it does, midway between the older conception of heart disease and the beginning of a new era of cardiac investigation, must of necessity adapt itself to both the old and the new *régime*, if it is to be of service to the practitioner of medicine.

The purpose, then, in writing this section, is to present to the physician those distinct clinical advances which are deduced from the highly specialized work being developed by the physicist, the physiologist, and the pathologist—to give place only to those cardiac drugs whose value has been established by recent investigations, rather than to burden our pages with remedies which have been administered empirically in the past.

The recently determined cardiac arrhythmias must take a place with the cardiac murmurs of the past generation in being considered only as *symptoms*, which may or may not call for any treatment other than supervision and watchfulness. Where other measures are indicated, the attempt has been made to correlate modern treatment with the modern conception of heart disease.

Through the courtesies of distinguished investigators, electrocardiographic curves and polygraphic tracings, which illustrate desired points, are presented. Thanks and appreciation are hereby extended to Drs. Alfred E. Cohn, of the Rockefeller Institute; Ross V. Patterson, of the Jefferson Medical College; Paul D. White, of the Massachusetts Gen-

eral Hospital, and Horatio B. Williams, of the College of Physicians and Surgeons, of New York, for the privilege of drawing freely on their store of valuable records.

The endeavor has been to make this contribution more practical than erudite, more helpful than argumentative, more suggestive than didactic—always bearing in mind what might well be called today's cardiac aphorism: *The muscle is of more importance than the murmur; the rhythm is of more importance than the rate.*

### CARDIAC IRREGULARITIES.

Irregularities of the heart, as shown by the pulse or by auscultation at the apex, have long been observed, and, through the usage of years, are generally believed to be indicative of heart disease. For example, in days past, children who presented arrhythmias have been regarded as potential heart cases, and their activities often so curtailed that proper physical development was interfered with; patients exhibiting an intermittent pulse have, in days gone by, been given guarded prognoses or perhaps refused as applicants for life insurance, and thereafter lived useful, energetic lives for twenty-five years or more. On the other hand, a slow pulse-rate of 50 which happened to be regular at the moment of examination was regarded as a "family characteristic," and the occasional "far-away" sensations which the patient experienced, or the syncopal attacks which he underwent at times, were attributed to indigestion or to epilepsy, with no thought of heart disease because no murmurs nor gross irregularities were present. Every practitioner of experience has noticed that digitalis, markedly effective in some irregularities, gave alarming symptoms in others; and the profession has been at a loss to correlate such opposed observations.

Today these irregularities are classified. We understand that digitalis is beneficial in auricular fibrillation, and often harmful in heart-block; we know that the arrhythmias of childhood are very often due to a change in vagal control, and that, alone considered, they are not pathologic; we appreciate that the intermittent pulse of adults is frequently due to premature contractions, and that, in itself, it does not constitute

heart disease; on the other hand, a pulse-rate of 50 or less is very suggestive of heart-block of some degree, and makes us alert in repeated examinations of the patient to determine whether such a condition threatens.

The masterful studies of Sir James Mackenzie, of London, enabled him to recognize and classify cardiac arrhythmias, giving us for the most part clinical signs by which we may be guided in their identification. The "ink polygraph" which he perfected, together with Einthoven's invention of the electrocardiograph, have, under the logical interpretation of Thomas Lewis, established a new era in the recognition of diseases of the heart. Indeed, the electrocardiograph might well be called *the quill with which the heart records the story of its own disease*. True, many deviations from normal are not yet interpreted and a vast array of research problems remains to be solved, yet results are continually being obtained promising notable additions to the brilliant clinical contributions already available.

*Cardiac Physiology.* A brief reference to the physiology of the heart is essential to the clear understanding of its irregularities.

The heart-muscle is possessed of five functions, viz.: stimulus production, conductivity, contractility, excitability, and tonicity. These functions may be disturbed, either singly or in combination, and give rise to cardiac irregularities. In the healthy heart, the stimulus for contraction arises at the sino-auricular node, situated at the junction of the right auricle with the superior vena cava. From there the stimulus is conducted along the auricle to the auriculoventricular node, from which springs the neuromuscular bundle of His, dividing in right and left branches, and conducting the impulse from auricle to ventricles. The excitation-wave is distributed along the arborizations of the branches. In disease, the stimulus to contract may arise from one focus or from multiple abnormal foci in the heart-muscle instead of from the normal "pacemaker," the sino-auricular node; the bundle of His may be damaged and refuse to conduct the stimulus; or the ventricle may originate stimuli of its own, quite independent of those received from the auricle. The polygraph and electrocardiograph, first used to demonstrate these departures from the normal heart



action, not only help in their recognition and treatment but have also given means by which many of the disorders may be clinically classified with a moderate degree of certainty, though the graphic method of heart examination should be used when available.

Let us for a moment follow the events occurring during a "cardiac cycle"—that period from the beginning of one complete contraction of the heart to the beginning of another. The stimulus for contraction arises in the "pacemaker," and is conducted along the auricle to the auriculoventricular node and over the bundle of His; the mitral and tricuspid valves now stand open, and the auricles contract in advance of the ventricles. The ventricles receive the stimulus from the terminal branches of the bundle at their base, and the ventricles contract. The mitral and tricuspid valves close. When the intraventricular tension exceeds that in the aorta and pulmonary artery, the semilunar valves (aortic and pulmonary) open and the *pulse period* begins. Ventricular systole completed, relaxation occurs, and when the intraventricular pressure is lower than that in the aorta and pulmonary artery, the aortic and pulmonary valves close and the sphygmic (pulse) period ends. The ventricles further relax, the cuspid valves open, the blood in the auricular reservoir passes into the ventricle during diastole, and a new cycle begins.

The cardiac cycle in a normal heart, beating at the rate of 75 times a minute, occupies 0.8 of a second. Of this time, 0.3 of a second is occupied by ventricular systole; 0.5 of a second by diastole, during the latter part of which auricular systole occurs. Increased heart-rate is almost entirely at the expense of diastole, with shortening of the period of rest. The period which elapses from the beginning of the auricular contraction to the beginning of the ventricular contraction normally occupies from 0.12 to 0.18 of a second; this is known as the A-s V-s (auricular systole to ventricular systole) period, or as the A-C interval in tracings of the venous pulse, and as the P-R interval in the electrocardiographic curves; any prolongation of this interval beyond 0.2 of a second, as determined by the time measurements on either record, is due to a delay of conductivity in the junctional tissues. In heart-block it may be prolonged to twice the normal interval.

*Instruments of Precision.* This article is not to be burdened by elaborating on the instruments for recording pulse tracings or electric curves; either subject, while most fascinating to those who can devote much time to its study, is intricate, difficult of mastery, and not suited to the purposes of the practitioner, in whose interest this section deals. Consequently, only a brief reference is here introduced.

*Mackenzie's ink polygraph* is so named because it records in ink, instead of upon a perishable smoked strip, the movements of the arterial and venous pulses. The apical impulse may be recorded instead of the radial. Radial tracings are called "sphygmograms" and tracings from the apex "cardiograms"; records of the venous pulse are termed "phlebo-

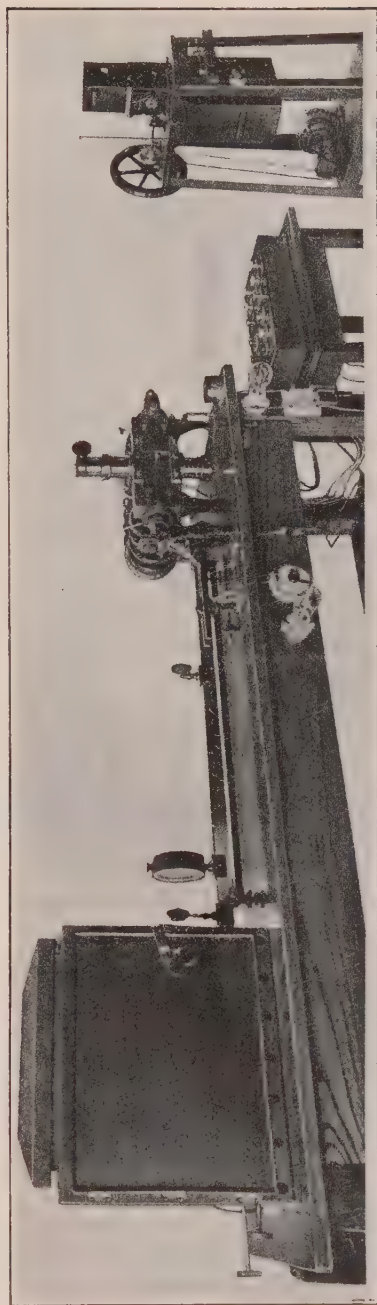


Fig. 1.—The Williams-Hindle American Electrocardiograph.

The outfit shown above is especially adapted for clinical investigation of the heart's action, inasmuch as its magnetic strength is three times that of other instruments so far adduced, the string does not "overshoot," the microscope does not jar out of focus, and the lamp retains its optical center.

grams." In using the polygraph, it is necessary to secure a tracing from the jugular pulse in order to establish the events which transpire in the right heart. A "time-marker" (the notched lines at the top of a tracing, 0.2 of a second apart) and "ordinates" (the vertical lines bisecting the tracings), from which to measure, as seen in the accompanying illustrations, are essential parts of the record. (See Fig. 9.)

The *electrocardiograph* (Fig. 1), or "string galvanometer," records upon a moving photographic film those electric currents generated during the contractions of the various chambers of the heart. The deflections of the delicate wire actuated by the heart currents vary in sequence, size, and incidence, and have been proved by Lewis to have normal limits in health, and any departure from these normal limits indicates an abnormal heart. The affection is perhaps in the auricle, in the ventricle, or in the bundle, as indicated by the deflections and by the time measurements recorded upon the film.

Positive and negative electrodes are applied in varying combinations to the arms and legs of the patient in order to establish the "leads" for the contraction-wave generated at the cardiac pacemaker which travels through the heart to produce its contractions. Three "leads" are usually taken from each patient, the second being the one which gives the most information, although leads I and III are needed for comparison and for further interpretations. Lead No. I is from right arm and left arm; lead No. II from right arm and left leg; lead No. III from the left arm and left leg. A set of normal electrocardiograms is here illustrated, the leads being denoted in Roman numerals. (See Fig. 2.)

*The classification of cardiac arrhythmias*, which we will now describe in the order named, is as follows:\*

- |                            |                              |
|----------------------------|------------------------------|
| 1. Sinus arrhythmia.       | 5. Auricular fibrillation.   |
| 2. Premature contractions. | 6. Heart-block.              |
| 3. Paroxysmal tachycardia. | 7. Alternation of the heart. |
| 4. Auricular flutter.      |                              |

---

\*Owing to the fact that atrioventricular rhythm, auricular standstill, ventricular escape, etc., are not yet recognizable by ordinary clinical means, but are to be detected only by graphic methods, their inclusion here would serve no useful clinical purpose.

1. *Sinus arhythmias* occur characteristically in the adolescent heart, and constitute the usual disturbances of rhythm found in the "youthful" irregularities. (See Fig. 3.) A famil-

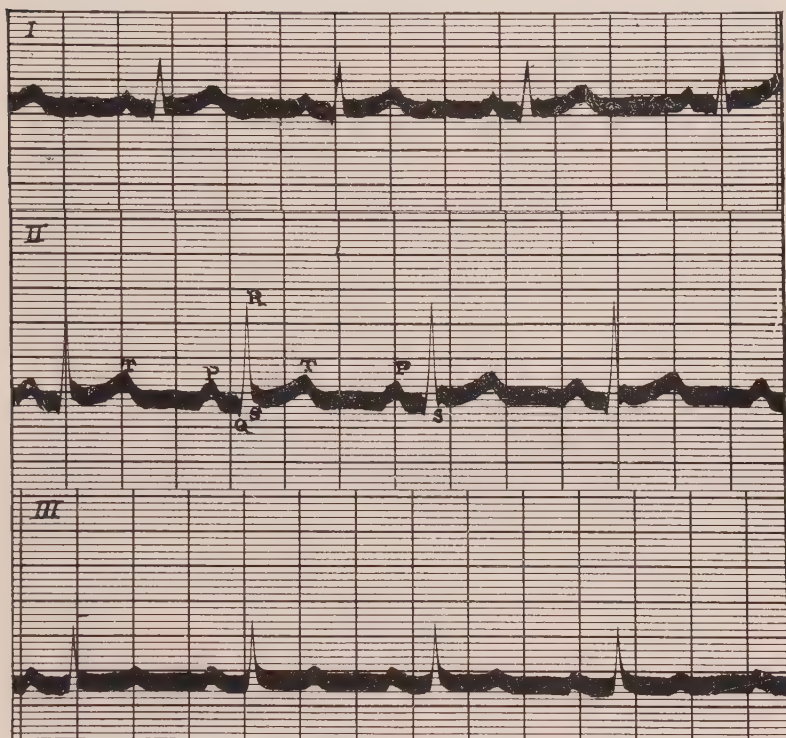


Fig. 2.—Normal Electrocardiogram.

Three leads are usually taken, for the purpose of comparative study. In this and subsequent curves the lead is denoted by Roman numerals in the upper left corner. The "abscissæ" (vertical lines) are time-marks, representing 0.2 of a second; the "ordinates" (transverse lines) measure 0.1 of a millivolt. The auricular contraction is indicated by the arbitrary symbol "*P*"; the ventricular complex is expressed by the letters *Q*, *R*, *S*, *T*. Diastole of the heart is the interval between *T* and *P*. *P*, a blunt-pointed elevation, is from 1.5 to 2 mm. in amplitude, directed upward. The interval from the beginning of the *P*-wave to the beginning of *Q* is the conduction time of the impulse from the auricular pacemaker along the bundle of His to the ventricles, and normally occupies from 0.12 to 0.18 of a second. In normal hearts, *R* is directed upward in all leads, usually attaining its greatest amplitude in lead II, where it varies from 10 to 20 mm. The *T*-wave, which expresses the end of the ventricular complex, is from 3 to 5 mm. in amplitude and is usually directed upward in all three leads. (This record is presented here through the courtesy of Dr. Paul D. White, of the Massachusetts General Hospital, Boston.)

ilar example is found in the change of rate in the heart of a young adult during the act of respiration, the rate being increased on inspiration and decreased on expiration. Unless



accompanied by other evidence of cardiac or other affections, it is not to be regarded as pathologic, nor does its detection indicate the administration of drugs or the curtailment of activities.

2. *Premature contractions* (also called extrasystoles) are exemplified in the "intermittent pulse," the abnormal beat occurring in advance of the anticipated interval, and usually being followed by a pause of unusual length. (See Fig. 4.)

Premature contractions are usually weak, while the succeeding and somewhat delayed beat is unusually strong, because of (1) prolonged ventricular rest, (2) relatively low intra-arterial pressure, plus (3) a greater accumulation of blood, all tending to increase the volume of the pulse. White, of Boston, found premature ventricular beats the commonest form of all disturbances of rhythm,<sup>1</sup> with the single exception of the semiphysiologic sinus arrhythmia just described.

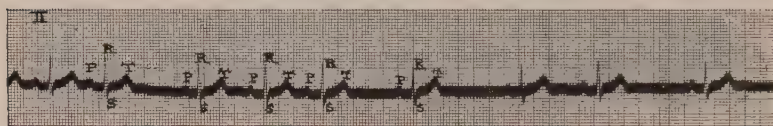


Fig. 3.—Sinus Arrhythmia.

This curve strikingly illustrates the variations in rate which characterize the "youthful type" of cardiac irregularity. The sequence of events is a normal P, R, S, T complex, but the rate varies with each contraction. (Courtesy of Dr. Horatio B. Williams, New York.)

Premature *auricular* contractions occur but one-third as frequently as those arising in the ventricle. Neither of these abnormal beats receives its stimulus from the pacemaker, but from an isolated and irritable abnormal focus. In the polygraphic record the ventricular premature contraction is distinguished by the fact that the two normal beats preceding the period of disturbance are of the same length as the abnormal beat and its predecessor; this observation is to be confirmed by finding that the "a-wave" of the jugular tracing occurs at the regular interval. Unaccompanied by other signs of cardiac disturbance, premature contractions have no great significance and require no treatment; the patient should be kept under observation, and examined at occasional intervals over a period of time, until it is established that the disturbance is unaccompanied by other evidences of cardiac disorder.



3. *Paroxysmal Tachycardia.* When the normal rate of the heart is replaced by a period of rapid and regular impulses varying from 120 to 200 per minute, which periods are *absolutely abrupt* in their inception and *absolutely abrupt* in their termination, paroxysmal tachycardia is present. The condition is often unaccompanied by other evidence of cardiac disease. Paroxysmal tachycardia is recognized clinically by a rapid heart-rate (usually from 160 to 180), which can best be counted at the apex rather than at the radial artery. Change of position from the upright posture to the prone does not alter the rate, and the abrupt onset and the abrupt termination of the attack,

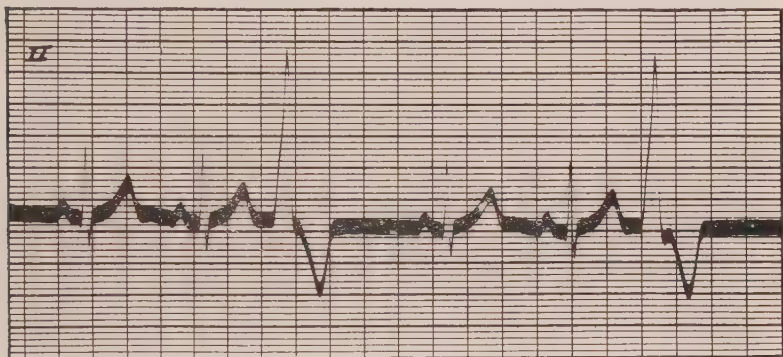


Fig. 4.—Ventricular Premature Contractions.

These produce a trigeminal pulse. Note the "tripling" of the ventricular complexes. (Courtesy of Dr. Paul D. White.)

whether it lasts for a few minutes or for a few weeks, are characteristic. One patient described the inception and termination as resembling "a mallet striking in the chest."

The prognosis depends upon the duration of the attacks, upon their frequency, and upon the signs of progressive cardiac failure, as shown in increase of the cardiac area, dyspnea, signs of congestion, edema, and cyanosis. The attacks may lead to exhaustion of the heart-muscle, hence prognosis will be guarded when the attack is long continued, despite the fact that infrequent paroxysms of but a few hours usually terminate favorably. Young patients may be assured that the attacks, while likely to recur at intervals, do not necessarily forecast a shortening of their life.

Pressure on the right vagus nerve, gently begun and gradually increased to a point where the carotid pulsations are obliterated, is effective in aborting perhaps one-third of the attacks. Vomiting may bring relief. A change of position may also be beneficial.

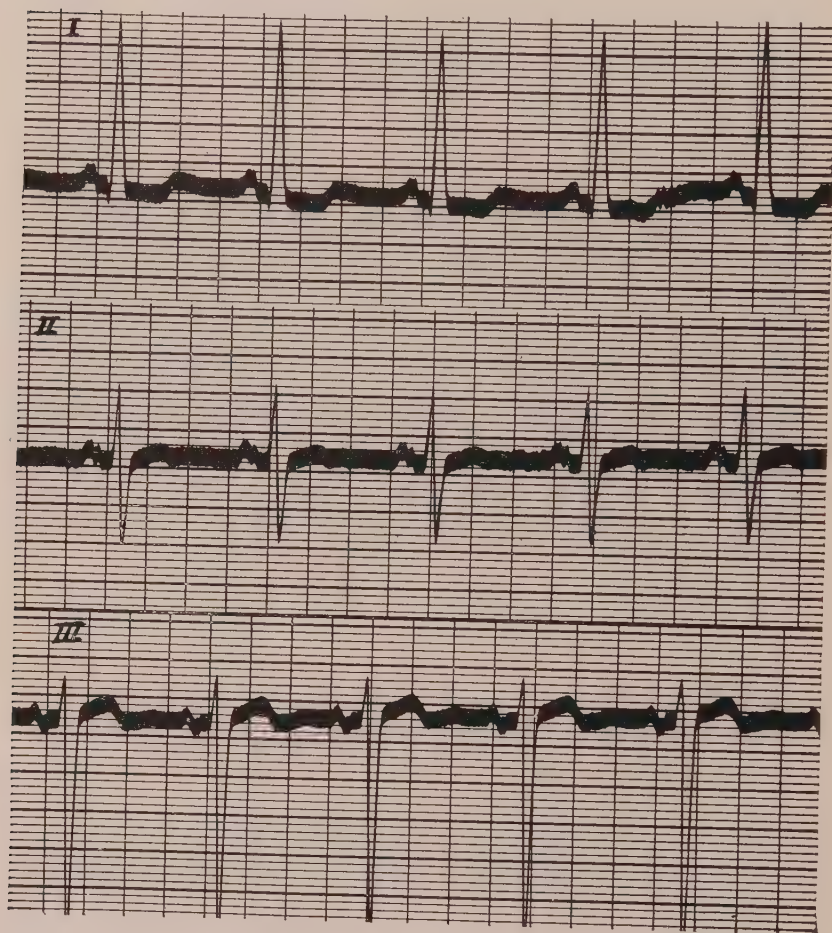


Fig. 5.—Marked Left Ventricular Preponderance, Showing Normal Sinus Rhythm.

The unusual amplitude of the *R*-wave in lead I, the marked *S*-depression of lead II and the short *R* plus the deep *S* of lead III, are indicative of left ventricular preponderance. These conditions are exactly reversed in the three leads when right ventricular preponderance is present. Compare with Fig. 7. (Courtesy of Dr. Paul D. White, of Boston.)

An ice-bag applied to the precordium is beneficial in many cases, and should always be tried. Not only physical and mental rest, but also the induction of sleep is called for. Morphin in the usual dose may be safely employed for discomfort or for insomnia. Venesection is rarely necessary to relieve engorgement and congestion. Lewis says that the "continued wearing of a broad abdominal belt, firmly applied before rising and discarded on retiring, is sometimes accompanied by the happiest of results."

4. *Auricular Flutter.* This is a term arbitrarily applied to a submerging of the normal auricular beats in response to a series of new, pathologic impulses varying in rate from 200 to 350 per minute (Lewis). (See Fig. 6.)

This rare condition differs from paroxysmal tachycardia in the fact that the enhanced rate is almost invariably associated

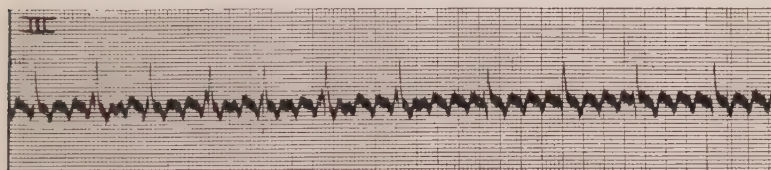


Fig. 6.—Auricular Flutter.

At the beginning of the curve the auricular rate is 315, the ventricular rate 105 per minute; every third auricular contraction is partly buried in the ventricular complex. In the latter portion of the curve the auricle contracts four times to each ventricular contraction. (Courtesy of Dr. Horatio B. Williams.)

with a failure of conductivity or contractility. From a single abnormal focus the stimulus for contraction arises, to drive the auricle at a regular and uniform rate. The affection is rare, and has its greatest age incidence in the sixth decade of life. In an elderly person who presents a persistent ventricular action of over 120 per minute, auricular flutter should be suspected and its presence confirmed by electrocardiographic examination. Another suggestive circumstance is the fact that there is absolutely no change in rate with change of position or after exercise in a tachycardia which persists for weeks or months. Strangely enough, the symptoms often consist of little more than a sense of fatigue; the profound constitutional disturbances that one would expect may be absent, except when the powerful ventricular muscle assumes response to

every auricular contraction, in which event signs of congestion, syncope, and cardiac failure supervene.

The condition is so recently discovered that nothing definite can be stated as regards the prognosis. Until such time as we have further light, the careful physician will be guarded in his prognosis and at the same time avoid giving the patient alarm with gloomy forebodings.

Full doses of digitalis is approved and efficient treatment. Following digitalis administration, auricular fibrillation often occurs, followed in turn by normal sinus rhythm. If the drug cannot be tolerated, full doses of strophanthin,  $\frac{1}{150}$  to  $\frac{1}{100}$  grain (0.0004 to 0.0006 Gm.), intravenously, may be substituted.

5. *Auricular Fibrillation.* This is a condition in which the stimuli for contraction arise, not in the normal pacemaker, but in multiple degenerative auricular foci. In this respect auricular fibrillation differs from the conditions previously considered, in all of which only a single irritable abnormal focus is at fault. The ventricular rate varies, usually being from 90 to 180; the pulse is wholly, continuously, and persistently irregular; in a succession of counts at the apex and at the wrist discrepancies will be found in practically every such count. Simultaneous counting of the rate at the apex and at the wrist shows a pulse deficit, *i.e.*, many of the ventricular beats are deficient (incomplete systoles) in reaching the wrist. Pandemonium reigns in the heart. (See Fig. 7.)

"Rheumatic" diseases of the heart are responsible for fibrillation in 66 per cent. of cases; whatever the cause, fibrillation of the auricles is most frequently associated with the classic symptoms of cardiac failure—profound dyspnea, visceral congestion, venous engorgement, edema, anasarca, etc.

Auricular fibrillation, in addition to the grossly and absolutely irregular pulse, presents a symptom quite apart from any other common cardiac irregularity, *viz.*, in fibrillation, exercise, emotion or fever (which raise the pulse-rate), make the irregularity all the more pronounced; in other common disorders of the heart-beat, exercise causes the irregularity to disappear. As the heart slows, the irregularity, in other disturbances, becomes *more* evident, but in fibrillation the irregularities become *less* evident. This variability of the



irregularity is of profound clinical significance. A rate of over 120 in a pulse that is absolutely irregular is almost conclusive evidence of auricular fibrillation. A presystolic murmur and thrill existing previous to the onset of fibrillation usually disappear when fibrillation occurs, owing to the inactivity of the auricle, maintained in a position of trembling diastole and failing to contract. This condition well illustrates the errors which constantly occur in estimating the ventricular

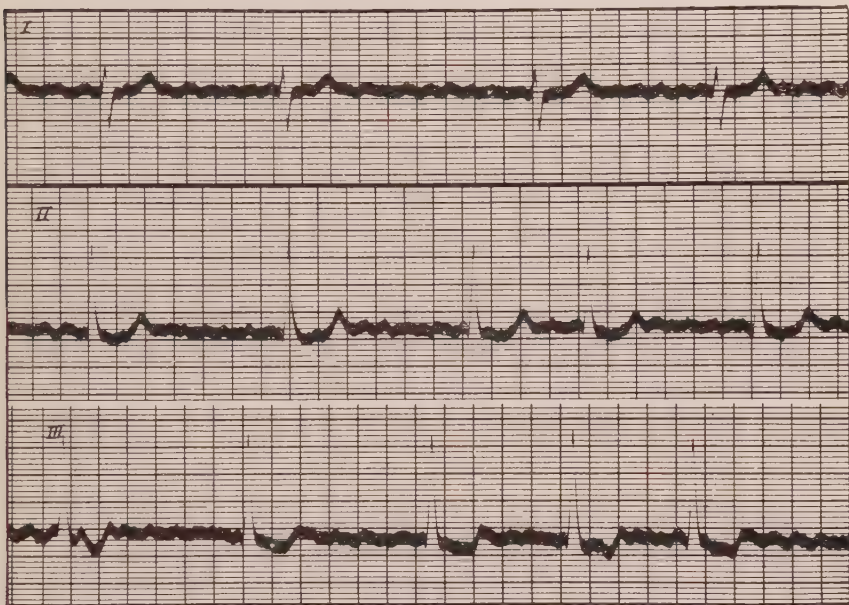


Fig. 7.—Auricular Fibrillation.

The ventricular rate has been slowed by digitalis. Note the substitution of fibrillary waves for the *normal* P-peak (as shown in Fig. 2). Note also that in this set of curves the R- and S- waves take a position in the leads the reverse of that in Fig. 2, thus giving us *right* ventricular preponderance. Further note in leads II and III, that the T-wave is "digitalized,"—that is, either decreased in amplitude or inverted; a change which has been observed within thirty-six hours following the initial administration of the drug, and which may continue for twenty-two days following its withdrawal. (Courtesy of Dr. Paul D. White.)

contraction by the pulse-rate alone; in fibrillation it is not infrequent to find a radial pulse of perhaps 64 and a ventricular rate of 130, showing that many of the beats fail to reach the wrist, and emphasizing the fact that the ventricular rate should be simultaneously estimated at the chest wall and at a convenient artery.



As may readily be imagined, the sounds are much altered in the tumultuous heart. When a beat is missed at the wrist, the first sound alone may be heard; when the beats are weak, so are the sounds; systolic murmurs, previously present, may be heard in fibrillation except when the rate is so fast that they vanish. (See Fig. 8.)

Fibrillation is an evidence of profound damage to the musculature of the heart. Fortunately it is amenable to remedial measures, and, while a serious symptom, is not by any means to be considered immediately fatal. If, in spite of therapeutic efforts, a ventricular rate of over 120 per minute is maintained, the outlook becomes progressively grave in proportion to the rate maintained. The converse applies—if a lower ventricular rate is maintained without the use of drugs, the prognosis is that much more favorable.

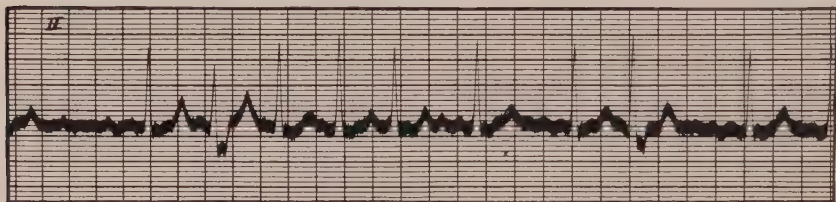


Fig. 8.—Auricular Fibrillation.

Showing an "ectopic" ventricular complex. An ectopic ventricular complex arises from an abnormal focus of impulse production within the ventricle; as the contraction does not proceed along the normal pathway of conduction in normally beating hearts, the rhythm is, of course, disturbed (Fig. 4), but here there is no rhythm to interrupt, hence the electrocardiograph affords us the only means of detecting the abnormal contraction. (Courtesy of Dr. Paul D. White, Boston.)

In the *treatment* of auricular fibrillation digitalis has achieved a brilliant reputation. Now that the condition can be definitely distinguished from other heart disorders, the early exhibition of the tincture in 10- to 15- drop (0.60- to 0.92- mil) doses four times daily is attended with results that are usually prompt and gratifying. When the heart-rate falls to 90, the dose is reduced, and when it approaches normal the drug is maintained at a dosage sufficient to continue its effect. The appearance of "digitalis coupling" (see Fig. 20), in which the beats occur in pairs or in triplets, a large beat being closely followed by one of smaller volume, is a signal for cessation of the drug. When urgent symptoms of fibrillation are seen, and the

heart-rate is above 170, strophanthin in doses of  $\frac{1}{250}$  grain (0.0002 Gm.) may be given intravenously, and repeated in two hours; a third dose is not frequently required, as the heart-rate falls to perhaps 90 within from six to twelve hours. Strophanthin, according to Lewis, is to be confined to the "rheumatic group" of cases in which the preferable drug, digitalis, occasionally produces symptoms of gastric disturbances. Venesection may be indicated, between 20 and 30 ounces (600 and 900 mls) of blood being withdrawn, to relieve the venous congestion.

The physician will exercise his judgment as to whether or not each individual case of auricular fibrillation shall be confined to bed, always remembering that cardiac exhaustion may rapidly develop at any time. Those who present more serious symptoms of cardiac disturbance and those of the higher heart-rate will, of course, be given the benefit of absolute rest, but milder degrees of the disorder may not require this cautionary measure.

It is to be remembered that belladonna and atropin are believed to increase the conductivity of the bundle of His, a condition which we absolutely wish to avoid in fibrillation. It is for the purpose of *decreasing* the conduction of the haphazard auricular impulses that we employ digitalis so successfully in this condition.

On account of the frequent association of auricular fibrillation with "rheumatic" diseases of the heart (which word is but a cloak for septic absorption from various foci of suppuration within the economy), this seems an appropriate place to mention that in *all* heart disturbances a search should be made for the hidden focus of infection. It may be found in suppurative ear or nasal-sinus conditions; perhaps the nidus exists in unhealthy tonsils; pyorrhea alveolaris may possibly furnish the infection; the appendix may act as a host for streptococci; gall-bladder infections or pyelitis may be unsuspected causes of septic absorption. But that cause so frequently at the base of septic absorption and so frequently overlooked is, in our opinion, apical abscess of the teeth. The physician should not be satisfied with the statement of the patient that his teeth were recently pronounced to be in healthy condition. A negative report from the dentist should not disarm the suspicion of

a dental cause when the conviction once has been established; only upon the receipt of a negative *x-ray* examination, in which the entire denture has been photographed, should abscessed teeth be excluded from consideration. The absence of large pockets of pus in the *x-ray* film does not exclude the teeth as possible foci of suppuration. Bliss<sup>2</sup> informs us that at the first examination the röntgenologist may find only some thickening or irregularity of the peridental membrane, but that an exposure made at a later date may show a slightly darker area around the apex of the tooth, indicating that absorption has taken place. Owing to the fact that dentists of earlier days did not fill the canal of the tooth down to and through the apical foramen, but proceeded to crown it, unmindful of this precaution which the *x-ray* has determined as absolutely necessary for safe results, capped teeth are to be regarded with



Fig. 9.—Partial Heart-block

The auricles are contracting regularly at the rate of 66 per minute. Stimulus conduction to the ventricle occurs every other beat, although the conduction is delayed (0.3 of a second); each alternate stimulus fails absolutely (block); the ventricular rate is, therefore, one-half that of the auricular (2:1 rhythm). The ventricular systole is 0.4 of a second in duration; diastole is 1.4 seconds in length, the departure from the normal rate being chiefly occasioned by an increase in the length of diastole. (Courtesy of Dr. Ross V. Patterson.)

suspicion, whether or not they give sensations of elongation, pain, or tenderness.

6. *Heart-block* is a condition in which the impulse conduction from auricle to ventricle is delayed or absent. The suspicion of heart-block should be aroused in heart-rates of 50; it is almost certainly present in rates as low as 35 per minute. (See Fig. 9.)

The transmission of the excitation wave from auricle to ventricle follows along the bundle of His. Mild grades of loss of function of the bundle of His are evidenced by prolongation of the A-s to V-s interval of 0.18 of a second. Slight grades of heart-block are evidenced by an occasional total failure of conduction, giving rise to a "dropped beat," in which







ventricular silence occurs for twice the normal interval. These may later recur at more frequent and regular (or irregular) intervals, as, for example, every eighth or tenth beat; succeeding upon this in more advanced degree we encounter a "2:1 rhythm," in which the auricle beats twice for every ventricular contraction; or a "3:1" or "4:1" rhythm may be established. (See Fig. 10.)

The extreme degree is that of *complete dissociation* of auricle and ventricle, in which no stimuli for contraction are transmitted; the ventricle may then be forever stilled, or it may initiate a rhythm of its own at a rate approximating 30 per minute. When ventricular silence extends over ninety seconds death results.<sup>3</sup>

When the ventricular silence lasts over three or four

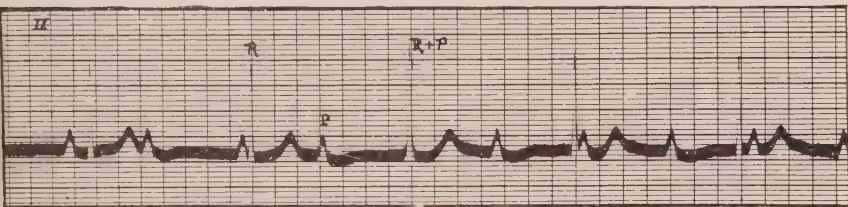


Fig. 11.—Complete Heart-block.

The *P-R* interval is normally from 0.12 to 0.18 of a second in duration. Here there is complete dissociation of auricular and ventricular contractions. Compare this curve with the normal sequence of events in Fig. 2. (Courtesy of Dr. Paul D. White.)

seconds, the *Stokes-Adams syndrome* may be manifest, in varying degrees of intensity. This condition, which is dependent upon an arrest of the blood supply to the brain, is characterized by slow ventricular and rapid auricular rates of contraction, attended by syncope, epileptiform convulsions, and visible venous pulsations in the neck, occurring more frequently than the arterial beats. The milder degree of this syndrome is characterized by a far-away sensation, perhaps by a feeling of dizziness or momentary loss of consciousness, and by muscular twitching.

The polygraph and the electrocardiograph afford the surest methods of determining the presence and the degree of heart-block, and the use of the instruments is essential for a diagnosis. Silence of the ventricle, rapid undulations of the

veins of the neck, the frequent (but by no means constant) presence of the Stokes-Adams syndrome, the possible halving of the ventricular rate, and a modification of the heart sounds, in which auricular contraction during ventricular silence may give rise to a third sound, are very suggestive clinical symptoms; taken in connection with a pulse-rate of 50 or lower, they furnish a symptom-complex which calls for the treatment employed in this condition. In very mild attacks, the patient is pulseless and momentarily pale, and in more severe attacks, giddiness, fainting, and temporary loss of consciousness, with muscular contractions, prevail.

Heart-block is to be regarded as an evidence of serious muscular change; it is not probable that this damage is limited to the bundle alone, but more than likely that the bundle shares in the general degeneration of cardiac tissues. Consequently, deaths from heart-block are deaths that arise from heart-failure, and not from any conditions peculiar to the block. So we must arrive at the prognosis in heart-block by estimating the amount of myocardial degeneration present, as shown by the severity and persistence of the usual cardiac lesions.

Patients with the milder grades of heart-block need not be confined to their beds, but may be up and around; even patients with the higher grades may attend to their usual duties, unless the signs of cardiac failure supervene. Treatment is directed to the underlying condition; if syphilis be the causative factor, antisyphilitics are employed. If "transitory" block arises in the course of an acute disease, such as the writer has observed in erysipelas, normal rhythm may initiate itself without *any* treatment. The failing heart muscle may require digitalis support, although its administration must be attended with extreme watchfulness and care, as the drug may convert an incomplete heart-block into complete block. If complete block be present, digitalis cannot make it more complete, and by its action on the cardiac muscle may prolong life. Atropin, by a possible effect of increasing the conductivity of the bundle of His, is the indicated remedy in all degrees of heart-block. It is given in doses of  $\frac{1}{100}$  grain (0.0006 Gm.), repeated as indicated for effect. Urgency may indicate the hypodermic use of the drug. (See Fig. 12.)

In heart-block produced in canines by forceps-pressure, epinephrin injections, according to the experiments of Heitz,<sup>4</sup> will restore auricular beats of 140 and ventricular beats of 55 to a common level rate of 115 within twenty seconds, but the effect soon is lost, and we await further evidence to establish the clinical value of the drug before advising its use.

7. *Alternation of the Heart.* This is a condition in which the pulse, regular in rhythm but varying in volume, alternates in

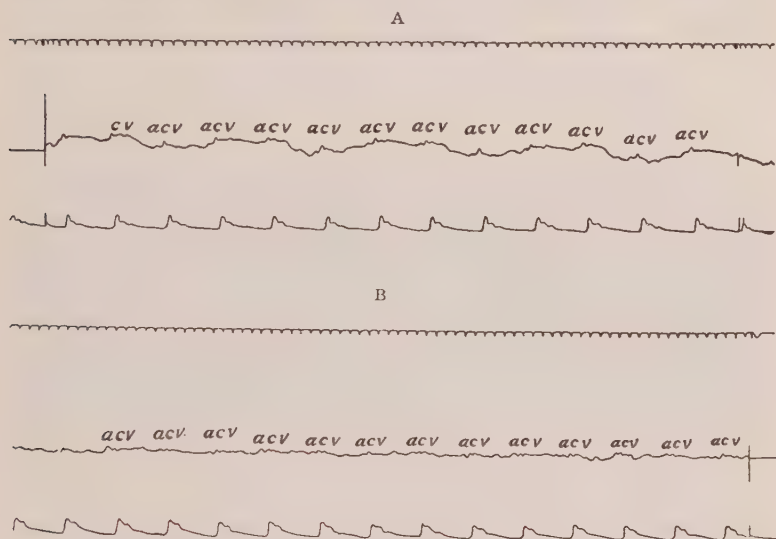


Fig. 12.—Effects of Atropin in Heart-block.

Same patient as Fig. 9, after the administration of atropin sulphate  $\frac{1}{80}$  to  $\frac{1}{50}$  grain (0.0011 to 0.0012) *t. i. d.* for several days. A twelve-minute tracing showed alternating periods of normal rhythm and 2:1 block of about forty seconds' duration each. The transition from one rhythm to the other was sometimes abrupt; at others irregular conduction caused arrhythmia for a few seconds. The section shown above (A) was from a portion in which no block occurred. Conduction from auricle to ventricle occurs in the normal time (0.2 of a second). The ventricular (and auricular) rate is 60 per minute. Systole is 0.3 of a second in duration; diastole 0.7 of a second. The atropin was continued and a few days later all block disappeared, and did not recur before discharge a short time thereafter, as shown in the tracing (B) which follows. (Courtesy of Dr. Ross V. Patterson.)

the height of every other wave. It is more frequently seen in advancing years in arteriosclerotic, nephritic, and anginal patients. We do not wish to convey the impression that the pulsus alternans is not encountered in acute infections and in the young, for it is associated with conditions which produce exhaustion of the heart-muscle, either acute or chronic. Alter-

nation is due to an impairment of contractility. (See Fig. 13.)

If the physician is to depend upon the detection of the pulsus alternans by the use of the examining finger alone, it will usually escape detection, for the alternate waves may be so small as to escape detection at the wrist. The sphygmogram, however, records their occurrence faithfully. In extreme cases the alternations appear in the entire length of the tracing. A clinical recognition of alternation can be established by placing the cuff of a blood-pressure apparatus around the arm and inflating it to that point where the weaker beats disappear at the wrist. Halving of the pulse-rate in this manner, while subject to error if the force of the smaller waves

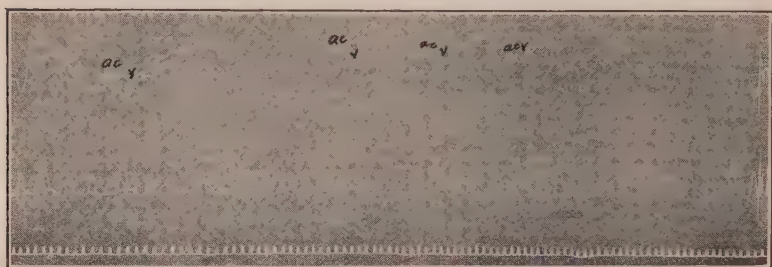


Fig. 13.—Alternation of the Pulse.

A very excellent example of *pulsus alternans* is shown. In two places the alternation of large and small beats is broken by the occurrence of two small beats. The small beats are incomplete failures of contractility on the part of the ventricle. The irregularity is one of volume only, the sequence being regular. *Pulsus alternans* occurs in conditions in which there is some serious affection of the myocardium force, and is of unfavorable prognostic import. The above tracing was made from a patient with evidence of fibrofatty myocardial degeneration and progressive cardiac insufficiency. (Courtesy of Dr. Ross V. Patterson.)

be comparatively high, or so faint as to be barely perceptible; should, nevertheless, be of clinical significance to the physician remote from graphic records.

The prognosis is that of the provocative condition. If acutely produced by toxic influences, in a heart that gave no evidence of previous affection, and if tending to disappear after rest, a pulsus alternans of infrequent recurrence is not of serious prognostic import. The majority of cases, however, are cardiopathic, and so associated with evidence of serious cardiac damage that the induction of the pulsus alternans is premonitory of the end. Frequently it may presage death

when Cheyne-Stokes respiration, angina, and other terminal symptoms are absent. Mackenzie found that the average length of life was two years following its detection. Of Paul D. White's 71 cases, 25 succumbed within ten months.<sup>5</sup>

The detection of alternation of the heart requires prompt change from an active mode of life to one that will conserve the output of energy and lessen the demand upon the weakened heart-muscle. It is to be borne in mind that physical exertion is not the only factor that imposes demands upon the heart, for psychic disturbances, emotion, and close mental application are no less drastic in their demands upon the weakened myocardium. That long periods of rest are indicated goes without saying; the diet must be easily assimilated, and limited in protein constituents; the emunctories of the body—the bowels, kidneys, and skin—kept in a healthily active state; and the occurrence of gastrointestinal disturbances, with their attendant heart load, vigorously combated. Digitalis in 10- to 15- drop (0.6 to 0.9 mil) doses *t. i. d.*, may be required to support the cardiac muscle, its administration being carefully watched for the untoward effects detailed at length in the discussion of this drug. (See p. 278.)

In the management of cardiopaths it is not enough that the physician content himself with the institution of a hygienic and dietetic regimen and with the instruction of the patient concerning rest. It is his duty to see that such instructions are obeyed, and that all sources of annoyance that might perturb an irascible patient are removed. As an illustration of the extremes of irritability common in men of dominant nature who are forced from the activities of leadership into a period of invalidism, we cite the instance of a venerable banker of our acquaintance. Naturally of a genial and cordial nature in health, he was, nevertheless, thrown into torrents of rage, which induced grave cardiac symptoms, when his nurse, in an adjoining room, used her handkerchief in a most unobtrusive manner. Trivial and unwarranted as was this cause of emotional disturbance, its immediate effect on the cardiopath was no less pronounced than would have been the news of a business calamity brought to him by the friends whom we excluded from the sick-room. The incident is mentioned here in the hope that it may be of service to others in searching for



the hidden cause of a failure to improve under circumstances apparently most propitious.

### PERICARDITIS.

Of the six affections of the pericardium, those which we recognize clinically are: (1) dry pericarditis (also called plastic or fibrinous, the "shaggy" heart); (2) pericarditis with effusion (either serous or purulent); (3) pericardial adhesions (adherent pericardium); the two last-mentioned frequently being gradually progressive steps, as a result of the fibrinous inflammation of the sac. The other occasional forms presenting a less definite symptom-complex are hydropericardium, hemopericardium, and pneumopericardium. The three last may be conveniently disposed of here with a brief consideration of each.

*Hydropericardium* is a transudate occurring as a part of a general anasarca, and presenting no physical signs by which it may be differentiated from an inflammatory effusion.

*Hemopericardium*, which results from a stab or other wounds that permit the ingress of blood into the pericardial sac, is of necessity rapidly fatal, and hence not clinically demonstrable.

*Pneumopericardium*, or the presence of air or gas in the pericardial cavity, has been recognized on less than fifty occasions in medical literature. The air enters through a wound of the sac, arising as a result of stab or gunshot wounds, of compound fractures of the thorax, of ulcerative processes extending from other viscera, or through a misdirected paracentesis of the thorax. The diagnosis is based on the presence of a splashing, churning, mill-wheel sound, such as might be expected from the movements of the heart within a sac containing fluid in the presence of air. The *x*-ray may adduce confirmatory evidence.

Before entering upon an extended consideration of affections of the pericardium, it may be well to recall the physiologic purpose of that membrane. Normally, Bernard has told us, it is an inextensible support for a dilating heart, and is capable of resisting an intense pressure of more than one atmosphere. This observation has recently been confirmed<sup>6</sup>

by experimental removal of the sac in animals, a procedure followed by a functional irregularity, by venous overdistension, and by incompetence of the mitral and tricuspid valves. Gradually the myocardium dilated to the point of rupture, first of the outer layers, but ultimately of the entire left ventricular wall.

Experimental distension of the pericardium by the introduction of oil<sup>7</sup> was followed by a steady rise of venous pressure, although the aortic pressure at first remained constant; but when the intrapericardial pressure rose to such a height that it prevented the filling of the heart during diastole, the supply of blood to the ventricles failed, the pulse became small, and the blood-pressure rapidly fell.

These experiments are of interest when one considers the amount of pericardial effusion frequently withdrawn clinically. A normal pericardium can hold less than 1½ pints (710 mls) of fluid, and yet amounts far in excess of this, even to the case reported by Gibson, where a gallon (4 l.) of fluid was withdrawn, are not incompatible with ultimate recovery. One is almost forced to conclude that the effusion occurring in pericarditis, up to an undetermined limit, exerts a beneficial, mechanically retarding effect on a heart affected by the toxins of disease; and the plastic pericardium, also, may by its inhibiting effect on a threatened heart, to some degree be considered as a manifestation of conservative effort on the part of nature.

Speculative as such a conception must necessarily be, the fact remains that pericardial effusions, naturally produced, rarely embarrass heart action; experimentally produced on an apparently normal heart of a dog, much cardiac embarrassment arises. Statistics are unavailable affording light on the mortality occurring in pericardial effusion treated by aspirating the fluid, as compared with the figures obtained by treatment other than surgical intervention.

Pericarditis, rare as a primary event, is usually associated with other diseases in which initial attacks of acute rheumatic fever hold first etiologic place. Its incidence is greater in proportion to the severity of the arthritis, according to Gerrod, and rarely exists with the milder "rheumatic" affections so frequently attended by endocarditis. Within eleven days

of the onset of arthritic symptoms, Gibson detected pericarditis in 50 per cent. of his cases. Chorea and tonsillar infections are common etiologic factors.

Other infections rarely induce pericarditis. It may occur in 3 per cent. of cases in pneumonia, and in less than 1 per cent. in scarlet and enteric fevers. Arising in the course of chronic conditions, it is frequently associated with Bright's disease and diabetes. Tuberculous infections of the pericardium are not rare. Pyemia may, of course, induce pericarditis, as may extension of an inflammation from contiguous organs or perforating wounds of the sac itself. Sex and occupation are irrelevant; a study of age incidence suggests that the pericardium should always be regarded with anxiety in the "rheumatic" affections of childhood.

Pericardial inflammation so often fails to reveal itself by either subjective or objective signs that its discovery is frequently accidental. According to Robey<sup>8</sup> this condition was clinically recognized only 100 times in 34,467 patients at the Boston City Hospital, and 12 times in 78 autopsies performed at the same institution. Attention is often first called to it as the result of detecting a to-and-fro murmur, produced by the friction movements induced by the heart in the inflamed pericardial membrane. This murmur, when present, is quite characteristic. Pain is of rare occurrence, and Mackenzie believes it to be present only when the myocardium is involved.

Pericardial friction may be palpated, but auscultation affords a better opportunity to make a differential study of this single physical sign in dry and fibrous pericarditis. On listening at the junction of the fourth left rib with the sternum, a grating, to-and-fro, superficial, leathery sound rarely bearing any relation to the cardiac cycle is audible. It is inconstant in intensity—now of greater, now of lesser volume; it may disappear for a few hours, depending on the strength of the cardiac contractions, and may be intensified if the patient be examined in a sitting posture. Pressure of the stethoscope may exaggerate the to-and-fro murmur. Pleural friction sounds need confuse us only long enough to instruct the patient to hold his breath at the end of expiration. Pericardial friction is then discovered still to be present, and to be intensified by this maneuver.

Upon the rapidity with which the effusion accumulates, and upon the quantity of the fluid, depends the recognition of pericardial effusion. It is manifest that rapid distension of the pericardial sac may produce urgent heart-symptoms; whereas, if the membrane has an opportunity to adapt itself to a gradual stretching, as in the case of chronic and slowly accumulating tuberculous effusions, cardiac embarrassment may not arise. Tuberculous fluid to the extent of a quart (1 l.) or more may be borne with surprising comfort on the part of the heart. Cabot states that less than 5 ounces (150 mls) of fluid are seldom recognized at the bedside.

*Inspection and palpation* reveal little of diagnostic significance; the third, fourth, and fifth intercostal depressions adjacent to the left of the sternum may be less pronounced than those of the opposite side. The cardiac impulse may be present when the patient reclines on the right side, for the reason that gravitation of the fluid in that direction causes the impulse to reappear; or the impulse may gradually disappear as the fluid accumulates. Palpation of the pulse with the view of detecting gross irregularities, or of discovering the "pulsus paradoxus" of pericardial effusion, is seldom of definite value.

*Percussion* over a cardiac effusion elicits an increased area of cardiac dullness, usually pear-shaped and extending as high as the second rib on the left. The dullness extends abnormally to the left, and on the right obliterates the right-angle dullness of the cardiohepatic junction. It is to be remembered that the effusion may change in location with a change in the position of the patient, giving us shifting areas of cardiac dullness. A dull percussion note behind, at the base of the left lung, first described by Ewart, is a valuable corroborative sign in differential diagnosis. By *auscultation* a progressive diminution in the intensity of the heart-sounds from day to day is noted, as the effusion interposes itself between the heart and the stethoscope. Compression of the adjacent lung provokes tubular breathing, increased tactile fremitus, and bronchophony in the compressed and atelectatic pulmonary area.

**Adherent Pericardium.** Systolic retraction of the tenth and eleventh left interspaces posteriorly—Broadbent's sign—is of diagnostic significance in adherent pericarditis. Smith's

sign is also of strong presumptive import; it is based upon the observation that the impulse of a normal heart has an excursion of one or two inches (2.5 or 5 cm.) as the patient lies upon the left side, and upon the further fact that the cardiac impulse descends during inspiration. Such excursions are not present in adherent pericarditis, and the cardiac impulse remains unchanged in position. Fluoroscopic examination by the Röntgen rays shows a restriction in the up-and-down movements of the heart accompanying breathing.

#### TREATMENT.

*Absolute rest*—physical, mental, and emotional—must be strictly enjoined, in order to insure the least possible degree of cardiac effort. The patient is permitted that posture in bed which is most comfortable to him. The writers are out of sympathy with the theory, advocated by a few, that early physical effort in pericarditis limits the area of pericardial adhesions by increasing the area of cardiac excursion. When one considers the arhythmias and muscle degenerations excited by increasing the heart's effort, the fallacy of such a theory is at once apparent.

Pain, usually due to myocardial inflammation, and not to the pericardial inflammation, frequently yields to the application of an ice bag or Leiter coil to the precordium; morphin in  $\frac{1}{4}$ -grain (0.0165 Gm.) doses hypodermically may be required to secure rest. In the stage of effusion, blistering of the precordium with two or three seasoned cantharidal plasters, cut in squares of  $\frac{1}{4}$  inch (6 mm.) each, often give gratifying results. The fly-blister is to be removed as soon as the vesicle forms.

Drug therapy is limited to that required by the causative condition. If acute rheumatic fever be the provocative infection, the salicylates are used in a daily dose of perhaps 60 to 120 grains (4 to 8 Gms.); the dose, however, is regulated by the effect secured, and may far exceed that here suggested. The salicylates are usually administered with sodium bicarbonate to lessen gastric irritation. Impending cardiac failure requires cardiac stimulants, and digitalis in supportive dose, 5 to 10 gtt. (0.31 to 0.62 Gm.) three or four times daily, may be indicated.



As to the surgical treatment of pericardial effusions, it is to be remembered that perhaps the majority are self-resolved under proper rest and treatment of the causative condition. When, however, no such tendency is shown and symptoms of cardiac embarrassment are added from day to day, surgical intervention is to be considered. Paracentesis of the pericardium, according to the statistics of Mignon, is followed by death in 65 per cent. of the cases, but in view of the absence of information as to how early in the disease this operation was performed, we should not permit these statistics to stay our hand in employing this measure for relief in urgent cases.

If the patient shows signs of oppression, and physical examination corroborates the view that a considerable effusion is present, the pericardium should be tapped and the fluid drained off. These symptoms are blueness of lips and fingers; rapid, small, compressible pulse; great dyspnea, pulmonary congestion, and the pulsus paradoxus.

The pericardium may be entered in one of several locations. Usually the needle is passed through either the fourth or fifth interspace, about an inch (2.5 cm.) to the left of the left border of the sternum. The right cardiohepatic angle has been advised by Rotch as the best site for the operation. Puncture may also be made in the fourth or fifth interspace to either side of the midclavicular line, or the third and fourth interspace, close to right border of sternum.

In any event, no matter what site is chosen, proceed with the greatest caution, bearing in mind two facts: one, that the diagnosis may not be correct; and, secondly, that even if the effusion be a large one, the heart may lie close to the chest wall.

In a case in the wards of the Pennsylvania Hospital, Le Conte removed 1700 mils (55 f3) of seropurulent fluid from the pericardium by means of a puncture made in the fifth interspace, just outside the midclavicular line. The proceeding was as follows: After cocainizing the skin, a small incision was made of size just sufficient to prevent catching the ridge of the trocar in the skin, an incident always calling for exertion of a little more force than otherwise would be needed, and at the same time often causing the sudden release of the instrument, and the consequent plunging it in more

abruptly and more deeply than necessary. For this reason the preliminary incision is particularly desirable in paracentesis of the pericardium. The trocar was introduced through the incision and carried into the pericardial cavity by means of a rotary motion combined with a cautious thrust.

Fluid should be withdrawn slowly, the patient watched for collapse, and, if it appear safe, his posture may be changed to the upright, in order thus to drain off more fluid. If signs of faintness and collapse appear, discontinue the operation and give a stimulant, such as camphorated oil hypodermically or aromatic spirits of ammonia by mouth. Under these circumstances, or even when all the fluid possible is withdrawn, it is sometimes necessary to make another tap because of its reaccumulation.

If the fluid does not become purulent, the case may get well slowly, but a certain amount of adhesions are likely to form, even in the absence of pus.

There is no remedy, either in the way of drugs, exercise, baths, or any other known procedure, that can prevent the formation of such adhesions, and their effects can be controlled in about the same measure. During convalescence, due consideration should be given to the likelihood of a more or less severe myocarditis having accompanied any case of pericarditis with effusion, grave or mild.

Following convalescence, prolonged rest and mental ease should be advised, and, for those who can afford it, travel.

### MYOCARDITIS.

The musculature of the heart may be subject to either acute or chronic inflammatory change. Acute infections of the endocardium do not always confine their activity to the lining membrane of the heart, but may readily extend through that delicate structure to the contiguous muscle-wall. While acute myocarditis and acute endocarditis arise as a consequence of infectious processes, yet they do not always coexist, nor, on the other hand, can a diagnosis of one be made without presupposing the coexistence of the other to some degree.

In view of the close relationship existing between myocarditis and endocarditis, the same factors are, of course, operative in the production of either condition. Bacterial invasion by staphylococci, streptococci, the *Diplococcus rheumaticus* of Poynton and Payne, the gonococcus, and the Klebs-Löffler bacillus are examples of the acute infections that induce myocardial damage. Influenza is an increasingly common cause of myocarditis. The specific fevers of typhoid, scarlatina, pneumonia, and the hybrid "rheumatic" group are to be carefully watched for evidence of cardiac implication. Conditions such as Graves's disease (thyrotoxicosis), which produce rapid and long-continued heart action, may, of course, induce cardiac exhaustion and subsequent myocardial change. Chemical poisons, among them alcohol, mercury, lead, and arsenic may precipitate acute myocarditis, or, when long continued, induce a gradually developed chronic myocarditis. The effect of the *Spirocheta pallida* upon the heart-muscle of the syphilitic should not be lost sight of as an etiologic factor.

The diagnosis of acute myocarditis is more a matter of deduction than a question of physical signs. When, in the course of a febrile condition due to bacterial invasion, we detect an irregularity of the pulse which heretofore has shown but the change of increased rate and volume; and when to this is added a sense of exhaustion out of all proportion to the severity of the infection, and otherwise unexplained, myocarditis may be suspected. The detection of fine râles at the base of the lungs, posteriorly, oftener occurring on the more dependent side, owing to the posture of the patient, is a valuable and early sign of myocardial inefficiency. Breathlessness, cyanosis, pallor, coldness, faintness, or intense prostration excited by the trivial exertion of sitting up in bed, confirm the suspicion. Any limitation of "the field of cardiac response" (Mackenzie) in either the bedridden or in those who are about the affairs of daily life calls for a prompt investigation of the heart. Precordial pain, or pain referred to the neighborhood of the first and second dorsal nerves, from which the heart, developmentally, receives its innervation, may furnish an additional guide to the recognition of the condition. Palpitation and tachycardia may ensue. An increased systolic pressure,

weak and irregular heart-sounds with occasional reduplication, systolic murmurs at the base, and evidences of cardiac hypertrophy, together with the history of an initial infection, furnish strong presumptive evidence of acute myocarditis.

The incidence of abnormal curves in an electrocardiogram occurring during the course of an acute infection in which there had previously been normal records is a diagnostic point of importance, and indicates the value of early and frequent electrocardiographic examinations during the progress of, and subsequent to, acute infectious disease.

The physician who fails to diagnose acute myocarditis correctly should not censure himself too severely for his lack of diagnostic acumen. The indefinite and variable clinical picture, often obscured by symptoms of the initial infection, may often cause us to overlook myocardial change. Cabot is responsible for the statement that 26 per cent. of cases of fibrous myocarditis are found *post-mortem* which had not been diagnosed during life; that 52 per cent. of the cases diagnosed during life were not found *post-mortem*, and that *in only 22 per cent.* did the clinical diagnosis and autopsy findings agree.

*Chronic myocarditis* may exist unrecognized for years and may be first observed when curtailment of accustomed activities draw attention to the heart. Perhaps one in middle life suddenly discovers that he is unable to spring up the staircase with his accustomed agility; he notices breathlessness and a sense of constriction about the chest as he hurries to his work; faintness and dizziness interrupts his game of golf; or extreme exhaustion and palpitation may follow the stimulation produced by motoring. With such mild limitations of the field of cardiac response does failing or broken compensation first manifest itself.

*Subsequent Myocardial Change and Prognosis.* Aside from those cases of myocarditis which, to all intent and practical purpose, recover, as a result of (1) cardiac resistance, (2) a limited degree of infection, or (3) through early recognition and skillful treatment, myocarditis is essentially progressive in nature. Rest, so necessary to a restoration of normal physical function, is a therapeutic measure obtainable only to a modified degree for the affected heart; complete rest is impossible. And so, as the heart labors on, cloudy swelling and granular degen-

eration may affect its musculature; implication of the coronary arteries may starve the cardiac muscle to such an extent that ischemic atrophy ensues. Hyaline and fatty degeneration are rare sequels to myocarditis; dissociation of one muscle-cell from another, each lying separate from its neighbor, has been observed in microscopic studies of myocarditis.

These conditions cannot be recognized clinically, and are not immediately incompatible with life. The heart, it has been determined by physiologists, is capable of putting forth thirteen times the effort normally required to maintain the body at rest, and this wonderful reserve power is not readily consumed. So while these muscular degenerations are not incompatible with life, they are incompatible with longevity, yet may permit an existence of cardiac invalidism until such time as complete exhaustion of the all-essential heart-muscle supervenes.

Fibrous increase of the connective tissue, usually distributed irregularly through the heart, is not at all uncommon. Fatty infiltration of the subpericardial connective tissue occurs very frequently, and its presence is often suspected during life in cardiac patients who exhibit a general tendency to obesity. Dyspnea and palpitation are symptoms of cardiac embarrassment in the obese; and muffled heart-sounds which, though faint, yet bear a normal relation to each other that is like a distant echo of themselves, have in the past been considered sufficient presumptive evidence for the once popular diagnosis of "fatty heart."

#### TREATMENT.

Emphasis should first be laid upon the value of securing as much rest as possible, by hygienic and dietetic measures, for the damaged heart-muscle. If the physician can secure a reduction of 12 beats per minute from a heart rate of 120, he has in twenty-four hours saved the laboring organ 17,280 cycles; in other words, he has given over four hours of additional rest (diastole) out of the twenty-four to the heart.

The indications for treatment are clear:

1. Rest—physical.
2. Rest—mental.
3. Rest—emotional.
4. Gentle elimination.



5. Nutrition of the heart to be improved.
6. Symptoms which add to the heart load should be relieved.
7. Drugs may be required to sustain the heart.

1. The patient should at once be put at *complete rest in bed*.

While this may seem too arbitrary a ruling for a given case, it is much easier to lessen restrictions as individual circumstances may require, rather than to impose added restrictions upon a patient whom we have permitted to be ambulant. As the symptoms improve, the liberty of the room or house may be allowed. The period of confinement to bed is usually continued until a normal rate and rhythm of pulse ensues, attended by the disappearance of other cardiac symptoms, with no unfavorable signs manifesting themselves on a change of posture. Judgment, of course, dictates that the aged and the infirm, who bear bed confinement poorly, shall be permitted a limited amount of liberty, of which limit they are often the best judges, rather than to be fretted and worried by the loss of appetite, insomnia, and mental depression attendant upon arbitrary curtailment of their mode of life.

2. That *mental rest* is of no less importance than physical rest is strikingly demonstrated by the beneficial results of sanatorium treatment. There the head of a household is freed from the worry of conducting or superintending the home, rid of the vexations of servants, away from the intrusions of oversolicitous friends, and is amid quiet, rest-inducing surroundings, where the ear is not strained to catch every unusual noise in the house, nor the mind kept busily employed interpreting each sound.

3. *Emotional disturbances* are difficult to guard against in the home-management of a cardiac patient, and they have a profound influence upon the heart. The attentions of well-meaning nurses may irritate; the sympathetic eye of a friend may cause depression of spirits; a whispered voice may be interpreted as ominous, or a laugh mistaken for an utter lack of feeling. In many such ways the emotions of a patient may be played upon, and, through the sympathetic nerve-fibers, actually affect the heart.

4. *Gentle elimination* is a term that is advisedly used. Mild laxatives, such as fluidextract of cascara sagrada or moderate

doses of the salines, used at frequent intervals, are to be chosen in preference to drastic cathartics which deplete the system and add the burden of physical exhaustion to the laboring heart. For the same reason, vigorous diuretics and the induction of free perspiration are to be avoided. A satisfactory diuretic in those cases where the non-employment of digitalis makes a diuretic advisable is:

℞ Spiritus ætheris nitrosi ..... f℥ss. (16.0 mls).  
 Syrupi ..... f℥ij (8.0 mls).  
 Liquor potassii citratis . . q. s. ad f℥iij (90.0 mls).  
 M. Sig.: Teaspoonful (3.75 mls) in a little cold  
 water at two or three hour intervals until effective.

The use of electric-light or other superheated cabinets may prove very exhausting to the myocardiac; the skin can usually be kept freely eliminative by tepid sponging, followed by witchhazel massage. The urgent incidence of uremia may, of course, demand hot packs; the occurrence of apoplexy will necessitate prompt and vigorous catharsis; but these are exceptional instances in the usual management of myocardial disease.

5. *Improving the nutrition of the heart* may be accomplished indirectly by easing the load, and by increasing the period of cardiac rest, as pointed out above. As to the direct effect of foods upon the heart-muscle itself, much yet remains to be discovered. The specious statement that "a diseased heart requires more nourishment than a healthy one" is at direct variance with the universal principles of diet in disease, where an effort is made to regulate protein intake as well as quantity of food. A cardiac patient who is at rest in bed or confined to his room manifestly requires less nourishment than when up and around; consequently, he must reduce the intake of food if he would avoid overloading kidneys, bowels, and liver, and by remote effect on these organs indirectly aggravate the condition of the heart and circulation. Small quantities of nutritious food at frequent intervals, the limiting of protein intake, and the elimination from the dietary of foods productive of intestinal fermentation in a given case are safer rules to follow than is the blind adoption of one of the many "cardiac diets." When it has been shown that these selected diets add length to the numbered days of the advanced heart

case, and when it has been shown that this increase of days was not attained by a sacrifice of appetite, and by proscriptions of dietary that made the extra days of life seem not worth while, then, and only then, can cardiac diets be said to have added to our therapeutics.

One notable exception to the general impracticability of rigid food-regulation in cardiac patients is the diet suggested by Karrell to be used when it is desirable to limit the *water intake*, which, by increasing an existing edema, ascites, or pleural effusion, adds to the embarrassment of the heart. An initial aspiration of ascitic fluid or of the pleural transfusion, should either be present, affords a degree of relief to the laboring heart, and the beneficial effect is maintained by instituting a diet which limits fluid intake to the nourishment found in milk; the free use of salt is interdicted for the reason that it accumulates in the tissues and attracts fluids to the parts. Even sufficient salt to maintain the normal individual demand of 15 grains (1 Gm.) per day is prohibited at first, until the excess previously stored in the tissues may be considered exhausted. The Karrell diet is as follows:

For the first seven days, 8 ounces (200 mils) of milk at 8 and 12 A.M.; 4 and 8 P.M. No other food or fluid.

Eighth day, milk as above, and at 10 A.M. 1 soft-boiled egg; at 6 P.M. 2 pieces of dry toast.

Ninth day, milk as above, and at 10 A.M. and 6 P.M. 1 soft-boiled egg and 2 pieces of dry toast.

Tenth, eleventh, and twelfth days, milk as above, and at 12 noon chopped meat, rice boiled in milk, and vegetables; 6 P.M., 1 soft-boiled egg.

No salt is used throughout the course. Salt-free toast and butter. Small amount of cracked ice with diet. All meat can often be advantageously omitted.<sup>9</sup>

*Cane-sugar in Heart Disease.* The classic experiment of F. S. Locke,<sup>10</sup> in which he demonstrated that the excised heart of a rabbit could be kept pulsating for four days by pouring through it a solution containing dextrose, has stimulated much interest in the clinical value of sugar as a means of nourishing depraved heart-muscle. Physiologists have discovered that the sinoauricular node, the bundle of His, and its arborizations, contain a remarkable amount of glycogen. Prof. Dr.

Adamkiewicz<sup>11</sup> states that the heart requires its own weight of sugar each day for its nourishment (9 to 11 ounces—254 to 312 Gm.). These observations have been utilized by Sir Arthur Goulston, of Exeter, in the treatment of heart disease with cane-sugar (he carefully avoiding the employment of beet and other sugars), yielding brilliant results. He begins with the administration of 2 ounces (56.6 Gm.) a day, pushing the administration rapidly to 4 ounces (113 Gm.) a day, or even to 10 ounces (283 Gm.) in some instances. The likelihood of digestive disturbances and intestinal fermentation is disposed of by the observation of Abderhalden<sup>12</sup> that the lactic acid ferment of the intestines does not attack cane-sugar or milk-sugar.

6. *Symptoms which add to the heart load*, and which the physician may have to ameliorate, are: anasarca, pain, sleeplessness, constipation, hepatic torpor, dyspnea, bronchitis, and vomiting. Where possible, remedial measures other than drugs should be employed. Dropsical effusions may be relieved by the trocar and cannula; painful engorgement of the extremities by Southey's tubes or multiple punctures ( $\frac{3}{8}$  inch—10 mm. deep) of the tense and edematous skin, always under antiseptic precautions and with subsequent aseptic dressing; pain may be amenable to the ice-bag or hot fomentations; sleep may be induced by hot drinks, by massage or friction rubs; constipation and hepatic torpor frequently yield to a diet of laxative foods or enemata; dyspnea, to a change in position, or the use of oxygen; bronchitis, to the gradual improvement of the cardiac condition; and vomiting, to a temporary withdrawal of all foods by the mouth, save the sipping of ice-water, and to the counterirritant effect of mustard plasters (1 part mustard, 5 parts flour) applied to the epigastrium.

7. *Drug therapy* in myocarditis, save for the emergencies arising in connection with the cardiac arrhythmias, is limited principally to the employment of digitalis and its congeners. The indication for its employment is either *a failure to improve in tone* or else *a progressive weakening* of the cardiac muscle when the simpler forms of treatment already outlined—rest, elimination, etc.—are ineffective. The large dram-a-day dose of the tincture is seldom required; 8 or 10 drops (0.5 to 0.62 mil) three times a day are quite often sufficient. The drug

will be withdrawn when the desired effect is produced. Digitalis, it is to be remembered, distinctly increases myocardial irritability and raises blood-pressure by slowing and increasing the force of the ventricular contraction.

Strophanthus and squill are cardiac tonics that are used when digitalis fails. Caffein, theobromin, and convallaria are employed in the milder cases, when a temporary change of drugs is advisable. The rapid, diffusible stimulant, aromatic spirit of ammonia, is at times of temporary service. Strychnin finds its best employment not in the treatment of the cardiac condition, but when combined with iron, arsenic, or quinin, in building up the general nutrition of the patient during convalescence. In this connection, while we hold no brief for any manufacturing pharmacists, believing that prescriptions should be, for the most part, specifically adapted to the individual case, we feel that we might well draw attention to a constant and dependable preparation of strychnin and iron, which latter drug is not permitted to oxidize during the process of manufacture, and which gives uniform results. We refer to "Tabloid Blaud," consisting of carbonate of iron, 5 grains (0.324 Gm.); arsenic  $\frac{1}{100}$  grain (0.00065 Gm.), and strychnin sulphate,  $\frac{1}{100}$  grain (0.00065 Gm.). The carbonate of iron does not react until it reaches the stomach, in strong contrast with many inert "Blaud" preparations on the market. The convenience of an iron preparation that is stable will be appreciated by the physician who is obliged to dispense his own remedies, as will also the form of administration, which guards against the definitely determined deleterious action of iron when it comes in contact with the teeth.

The  $x$ -ray has a therapeutic value in myocarditis, due to excessive glandular activity, such as is exemplified in Graves's disease, or exophthalmic goiter, the Röntgen light reducing the activity of the gland, and thus lessening the quantity of toxins set free to act injuriously upon the heart.

### ENDOCARDITIS.

Inflammation of the lining membrane of the heart arises as a complication of, or as a sequel to, acute infections occurring within the body. On account of its frequent association with the symptoms of septic absorption, and as a result of the ob-



servations that the condition more usually occurs as a sequel to rheumatic fever, tonsillitis, and chorea, the misleading term "rheumatism of the heart" is sometimes applied to endocarditis. The term should be discarded. It gives no clear conception of the condition, and may utterly misdirect the treatment of the physician who relies upon "antirheumatic drugs" to correct the perverted cardiac condition.

When one considers the influence of febrile conditions upon the circulation, which frequently raise the rate from 8 to 10 beats a minute for each additional degree of fever, the probability of infection of the overworked heart by disease germs already actively engaged in elaborating toxins in other parts of the body become quite manifest. Among the bacteria detected in the endocardium at autopsy are streptococci, staphylococci, the *Diplococcus rheumaticus*, gonococci, Klebs-Löffler bacilli, and the bacillus of Koch. The condition may also arise in connection with specific fevers, such as pneumonia, scarlatina, diphtheria, and enteric fever.

Depending upon the virulence of the initial disease, upon the resistance of the heart to infection, and upon its ability to withstand the labor imposed upon it, acute endocarditis may develop into the *malignant* form. There is no well-defined line of demarcation between the two; nor can too fine a distinction be drawn between that point where either variety evolves itself into chronic endocarditis (valvular disease of the heart); the difference is but a difference of degree. It should be borne in mind that we are not always able to trace a history of antecedent infection, and that acute endocarditis may seem to arise as the consequence of an infection of the heart alone, with apparently no other organs implicated.

The left heart presents the more constant pathologic change, the area of the mitral valve showing endothelial degeneration; next in frequency the aortic cusps are infected. Roughened valve-surfaces become the seat of fibrin deposits whipped from the blood-stream, these vegetations frequently being infected and swept as emboli to other parts of the body. Coincident with thickening of the cusps, or with the formation of vegetations, retraction of the leaflets may occur, giving rise in life to the murmurs which often first direct attention to the presence of endocarditis.

Morbid changes do not invariably affect the valves; they are not confined to the endocardium alone; the myocardium and pericardium may be affected, constituting a pancarditis. Especially is this true in the streptococcic heart affections of children.

This disease presents no definite clinical signs by which we may detect its presence. Its recognition depends upon



Fig. 14.—Malignant endocarditis, with extensive implication of aortic valves (Philadelphia General Hospital). (From Da Costa's Physical Diagnosis. Copyright, W. B. Saunders Co.)

the astuteness of the medical attendant who correlates the variable symptom-complex; the diagnosis is rarely found in the admission records of hospitals. The suspicions of the clinician are aroused when, in the course of an acute infectious disease, he detects irregularity in the volume and rhythm of a pulse previously having shown no variance other than an

increase of rate. An elevation of temperature unusual for a given disease, or showing a persistence beyond that ordinarily encountered, and which cannot be otherwise satisfactorily explained, should call for detailed and frequent examinations of the heart. A leucocytosis out of proportion to an existing arthritis, tonsillitis, chorea, or a persistently high leucocyte count following these conditions, may point to endocarditis.

The occurrence of cardiac murmurs where none had previously existed, and the tendency of these murmurs toward daily variations of both location and intensity, are most significant. Usually they are heard at the mitral area, and are systolic in time, indicating regurgitation; during the period of valve-roughening they may be of a musical quality. Mitral murmurs are observed with marked frequency in choreic patients. Concomitant hypertrophy of the heart may exist. The occurrence of embolic occlusion of an artery is of diagnostic significance in endocarditis.

The less complicated form of acute endocarditis, which does not develop to the degree of malignancy, is usually not fatal. The patient may recover entirely, or receive the heritage of a "damaged heart," which eventuates in chronic valvular disease. In the malignant form, where the lining membrane of the heart shares its damage with the myocardium, or where septic thrombi are whirled to other portions of the body, the outcome is a question of profound gravity.

#### TREATMENT.

It is of vital importance that *absolute rest in bed* be insisted upon. The patient with either acute or malignant endocarditis is not permitted even to turn himself from one side to another, until the symptoms subside; bed-pans and urinals save physical effort and thus conserve cardiac strength. When we reflect that an affected heart which makes 8 extra beats a minute makes 11,520 cycles a day more than are required in health, we can then appreciate the absolute necessity of sparing the organ even such a demand as would be occasioned by the effort of extending the hand. We should remember, too, that increased heart-rates are usually at the expense of the rest-period of each cycle. More effort means more exhaustion; more exhaustion means less resistance of the cardiac

tissues already affected, and a consequent extension of the inflammation.

The physician who presents the necessity of rest to a patient in a manner that causes alarm is temperamentally unfortunate. Instructions should be given to the nurse, thus sparing the patient the added burdens of anxiety, alarm, or depression.

The hygienic indications of elimination are met as required. Gentle catharsis is secured by fluidextract of cascara sagrada in 10- to 30- drop (0.6 to 1.8 mil) doses, effectively meets the indication of peristaltic stimulation, and avoids an unwished-for depletion of bodily fluids with the larger and sometimes nauseating doses of salines. The nurse will employ tepid baths, followed by witchhazel rubs, to keep the skin in active condition; the kidneys are stimulated with small but frequently repeated drinks of water to which fruit juices may be added. Diet is of the form most readily assimilable and is free from any substance which, by provoking indigestion or fermentation, may add to the load of the heart. Massage, if employed in the acute stage, must be most intelligently directed; pain and coldness in an extremity, arising as a consequence of an embolus having lodged in the peripheral circulation, may cause a well-meaning nurse to massage the part with no thought of the possibility of friction breaking up and further distributing the infarct. Heat may be employed to relieve the sensory disturbances caused by an embolus; opiates may be required for pain. Insomnia, which is the bitterest antagonist of our much-sought-for rest, should be controlled by quiet surroundings, well-ventilated chambers, and by the employment of opiates in sufficient dosage to secure the results desired.

The next step in treatment is directed to the removal of the underlying cause. If acute rheumatic fever be the provocative infection, the salicylates are employed in 10- to 20-grain (0.66 to 1.32 Gm.) doses at three- or four- hour intervals—always sufficiently diluted, and always combined with equal doses of sodium bicarbonate, for the purpose of preventing gastric irritation, and in the hope of preventing the possible occurrence of salicylate-poisoning by thus rendering the urine alkaline. The physician may have to abandon the use

of the salicylates in order to spare the stomach—a step which he will promptly take upon the appearance of gastric irritation or other evidences of disordered digestion.

Chorea is treated by the administration of *liquor potassii arsenitis* (Fowler's solution), beginning with 3-drop (0.18 mil) doses, well diluted in water, *t. i. d.*, *p. c.* The dose is increased 1 drop (0.06 mil) at each administration, until the physiologic limits of arsenical tolerance become manifest by slight puffiness under the eyes, looseness of the bowels, and griping. When these symptoms occur, the drug is withdrawn for a day or two, and its administration again begun in a daily decrease of dose until a minimum of 5 drops (0.31 mil) *t. i. d.* is reached, and then increased as before.

Autogenous vaccines (*i.e.*, vaccines derived from that particular variety of germs present in a given case, as determined by bacteriologic examination and cultures from the blood, causative abscesses, etc.) may be required; and when used should be used early. Laboratory delays in securing an autogenous vaccine may require the employment of "stock" (already prepared and marketed) vaccines until the more desirable autogenous serum can be obtained. As to the efficacy of this form of treatment much doubt exists—a doubt which entitles the patient to the benefit occasionally reported in endocarditis from its employment.

Further steps in the treatment of endocarditis arising independent of specific fevers (which are treated according to the indications for each), of course, embrace an unremitting search for suppurative foci in tonsils, at the apices of teeth, in ear involvements, gall-bladder infections, perirenal abscesses, cutaneous affections, cystitis, pyelitis, and bone or joint involvements.

Cardiac weakness may require tincture of digitalis in 5- to 10-drop (0.31 to 0.62 mil) doses at four- or five- hour intervals. It is not to be used as a routine, but only to meet indications of cardiac muscular weakness or compensatory failure.

*Convalescence.* The duration of the rest-period in those patients who have passed through acute or malignant endocarditis is one of profound importance. It is to be continued until all symptoms have disappeared, and until the rate and



rhythm of the pulse are normal. The patient may then be permitted to sit up for a brief interval, which is gradually lengthened as the days pass, and as no unfavorable change in the area of cardiac dullness or rate and rhythm of the pulse ensue. This usually means six or eight weeks in bed, and another month on a couch, before any activity is resumed.

The physician who thoroughly appreciates the pathology of the soft and infiltrated valves in acute endocarditis, and the possibility of resolution terminating in scar-formation on the leaflets by ill-advised exertion, will hesitate to assume any responsibility for the probable chronic valvular disease which may be caused by a disregard of the essential element of rest.

### VALVULAR DISEASE.

**Clinical Pathology.** *Rate of Heart-beat.* Under normal conditions the pulse-rate and heart-rate are equal, and the beats occur at about 72 per minute in an adult.

The rate is influenced by exertion, food, certain kinds of drinks, such as coffee and alcohol, and by various diseases. Fevers, certain toxic states, such as Graves's disease, chronic valvular and myocardial change, may and do increase the rate above the normal.

The rate may be persistently above normal, or may be so only at intervals; and a different significance attaches to the symptom because of the constant or fugitive quality of the change.

Thus, a persistently rapid rate of the heart-action suggests, in the absence of any of the causes just enumerated, a chronic valvular or myocardial change, and a heart that is trying to make up by haste for what it lacks in power.

An occasionally rapid pulse, particularly if it be very rapid, suggests a change of a rather inexplicable nature associated with paroxysmal tachycardia and auricular flutter or fibrillation, which conditions are dealt with more fully under Arrhythmia (*v.s.*). A too rapid pulse after slight exertion, and a failure to return to the normal rate within a short time, afford to a slight extent an index of the cardiac reserve force.

A *slow pulse* is more uncommon than a rapid one, and yet there are occasional instances of slow pulse consistent with health.

*Slow pulse* is seen occasionally in brain tumor, meningitis, and jaundice. It is also met with in cases of aortic stenosis, although by no means is this a constant occurrence, and in certain forms of cardiac disturbances, where the pulse-rate is not a true indication of the heart-rate.

For instance, in cases in which the auricular beats may be very frequent, many of the impulses are not conveyed to the ventricle; and again a certain number of these impulses may be responded to but feebly by the ventricle, with the result that no pulse impression is carried to the palpating finger.

Premature systoles may cause an apparent slowness of the heart's action, as shown by an examination of the pulse alone, while heart block in which the auricles and ventricles are beating with little or no relation to one another, causes a real slowing of the ventricle.

**Valvular and Muscular Disease.** It is rather a common view that unless there are very evident signs of disease of the heart there is no serious ailment. The presence of a loud murmur, signs of great enlargement of the heart, pulsation of the precordia and vessels of the neck, and attacks of angina or general anasarca are alone the indications of gravity in heart cases, according to views held more or less generally.

It is necessary, however, in order to arrive at an intelligent prognosis, to consider a great many other factors, some of which are at first apparently but trivial, but which, when considered together, and dovetailed into proper relation with the history of the case, may prove of greater value to a correct understanding of the state of the heart and its reserve force than the presence of the more evident and more easily recognized signs.

Valvular disease may or may not be serious, according to the condition of the heart-muscle and its reserve force, which latter may be regarded as an individual attribute, and therefore hard to compute in concrete terms.

The effects of a diseased heart valve are too well known to need much explanation in detail.

The diseases of the valves give rise to stenosis or regurgitation, as the case may be, or to both conditions in one and the same valve.

To overcome this defect in orifice or valve, and to keep up the circulation and supply the needed nutriment to the tissues of the body and the heart-muscle itself, the organ is compelled to do more work than it does under normal conditions.

This is accomplished in most cases in a greater or less time by the development of dilatation and hypertrophy. Dilatation of a chamber of the heart is a necessity in overcoming the diminution of the amount of blood in circulation as a result of a valvular lesion. In other words, if there



Fig. 15.—Comparative sizes of the ventricles in a normal and a hypertrophied heart (Philadelphia General Hospital). (From Da Costa's Physical Diagnosis. Copyright, W. B. Saunders Co.)

is a back leak in a valve, a greater amount of blood must be held in the propelling cavity to compensate for the inevitable loss when the contraction takes place.

Granted that this be true, hypertrophy becomes a natural corollary in a muscularly healthy heart, the muscle tissue enlarging to do the extra work of discharging from the cavity a greater amount of blood than normal in practically the same time as is granted the normal heart to discharge its lesser quantity.

In the event that a ventricle—for that is the cavity most commonly affected in valvular disease—is not able so to take

care of a lesion of the valve, an explanation may lie in the fact that the heart-muscle itself may have heavily shared in the attacks upon the endocardium, and been rendered unfit to carry on its part of a handicapped function.

There is a great difference between this kind of dilatation and that which comes on in the course of a chronic heart lesion, associated with dropsy and the long train of symptoms connected with general heart-failure.

Mackenzie, taking as a basis the functions ascribed by Gaskell to the heart-muscle, makes a strong and convincing argument that dilatation of the heart—a cause of heart-failure in the valvular cases—is due to the failure of one of the particular muscle functions, namely, tonicity.

For instance, he quotes cases where the muscle tissues of the ventricle had become so worn and thin that rupture had taken place, and yet neither before nor after death was dilatation apparent. And with these instances account must be taken of high pressures and constant effort, even to the rupture-point; but the mystery of the function of tonicity remains quite as dark as the formerly accepted theory of muscle exhaustion.

There are instances of sudden cardiac failures and deaths in those who show no muscular nor valvular heart disease, where certainly none of the phenomena that are part and parcel of the customary changes incident to cardiac failure according to the exhaustion and back pressure theory are evident.

At the same time no one will claim that failure of an important muscular function of the heart takes place in a heart-muscle free from disease or exhaustion, and certainly our inability to demonstrate such change is because of ignorance and not because of the lack of evidence.

The action of the heart, according to the recent views, is dependent upon the integrity of the cardinal functions, already spoken of, and to the co-ordination of these functions as well. It is likely that a failure in one of these functions will upset the others to the extent of causing serious heart embarrassment or death.

The generally accepted view of the progressive steps leading to cardiac failure is much as follows:

The leakage from a valve causes dilatation and hypertrophy; the extra amount of work and strain thereby provoked in the effort to keep up circulation leads to the hypertrophy and dilatation of the chamber next adjacent, and so on until the right heart and tricuspid valves fail, with the appearance of congestion of the liver and general dropsy.

At any rate, whether or not the change be directly due to muscular exhaustion, to back pressure, or to failure of an arbitrarily assumed function of the cardiac muscle, dilatation is usually associated with heart-failure as a cause.

The treatment of heart conditions arising from damage and partial destruction of a valve or of several valves, in no wise differs from the treatment of heart-failure from other causes except as regards the etiologic factor at work, which to some extent may be controlled.

The fact of the existence of a heart-murmur, the evidence of a valvular disease, does not *per se* call for any treatment whatever, but rather for advice as to the proper mode of life to be pursued in order to preserve to the utmost the reserve force of the heart, and to make so little demand upon it as to leave the patient with a comfortable margin in time of need.

As has been said before, the treatment of any condition of heart-failure must be based upon a study of the muscular condition, and must be directed with a view to resting, conserving, and relieving the strain or infection from which this muscle suffers. It must be borne in mind that overtreatment may do harm in heart disease as in other states, and that it is only when there are clear indications for treatment that any drugs or other methods should be employed.

As intimated above, there is some difference in the treatment from the point of view of the etiology. Thus, the treatment of a heart condition resulting from an acute infective process, such as rheumatism or septicemia, naturally differs from that due to an active syphilitic process, as in each case the cause of the disease demands active specific treatment.

This, however, is largely aside from the question of the treatment of the actual heart-failure itself, and here, whether this be due to valvular disease caused by rheumatism, or to sclerotic processes of old age and syphilis, the problem is the same, and is centered in the muscular tissues.



**Pathologic Physiology of Valvular Disease.** Without going into the question of the minute pathology of endocarditis, it is sufficient here, for the purposes of rational and understanding treatment, to consider the results of the inflammation of the endocardium present and remote.

A large percentage of endocarditis—almost all cases in the young—are due to rheumatism, chorea, and, to a less degree, scarlet fever. Those diseases confined to the aortic valve in adult and later life are due to syphilis and sclerotic processes of advanced life. Many authors hold that hard physical labor is a frequent cause of aortic disease.

Curiously enough, a great many cases of mitral stenosis, even in the young, give no history of any of the infectious diseases so common in their association with valvular heart disease.

Endocarditis, be it from what cause it may, causes a thickening, roughening, and shrinkage of the valves, and in some cases a sclerotic change at the orifices, which result in a narrowing of the opening affected. This latter condition, spoken of as stenosis, or obstruction, is often a progressive lesion, advancing either with moderate rapidity or very slowly indeed. In the case of stenosis of the mitral orifice, where it has become extreme, the valve as such ceases to exist, and in its place is found a diaphragm formed by fusion of the leaflets covering the whole auriculo-ventricular orifice, with a mere slit for an opening, and where from the nature of things it is apparent that no valve function any longer exists.

Whether the endocarditis and resulting changes be of the nature of a regurgitation or an obstruction, the heart at once suffers from a handicap, and is obliged to do more work in a given time than formerly, and to do this work dilates and hypertrophies according to its strength and nutrition, and the result is what is commonly spoken of as a compensated heart.

By this we understand that the heart has sufficient reserve force to respond to the needs of the circulation, although called upon for more than usual exertion.

For the time, then, the patient is to all intents and purposes as well as a person with healthy valves, save for the fact that he is not able, or could not be able for long, to stand

as much work as the healthy man, because of the necessary encroachment on his reserves.

There are many factors that have to do with the length of time a heart disease caused by a valvular lesion may continue without causing symptoms.

One thing, however, is certain, and that is that no drugs will benefit the valve itself, and that the patient will not outgrow the disease. The valve disease will last as long as the patient; and in most cases, unless an intelligent mode of living be adopted, the disease will rapidly outgrow the patient's strength and reserve force, and, in the case of the young, consign them to an early grave.

The question of the ultimate breakdown of the heart and the consequent heart-failure has already been gone into at some length under the caption of Myocarditis. (See p. 228.)

Whether this heart-failure be due to back pressure or to failure of tonicity makes no difference as far as the treatment is concerned.

The actual degree of incompetence or of obstruction must be considered in determining the probable duration of life of a patient suffering from valvular disease; also the mode of life; temperament; the susceptibility to rheumatism, tonsillitis, etc.; the infecting cause; the ability to control syphilis or arteriosclerosis, if present; and the personal equation of the patient's inherent strength and resistance, formerly spoken of as his constitution.

So far the valvular diseases have been spoken of as a whole rather than as individual conditions, because of the fact that the failing heart-muscle, and not the valve demands treatment. As regards prognosis, however, there is a difference in respect of the valve or valves affected.

Simple mitral regurgitation may be regarded as the least serious of valvular lesions, and aortic regurgitation and mitral stenosis, in their order, next.

Tricuspid disease is rarely seen except as a condition secondary to one of the other lesions, while pulmonary disease is almost invariably congenital.

Aortic regurgitation is most frequently associated with sudden death, often when the patient has been entirely unconscious of the existence of any cardiac trouble whatever.

This it is that makes it imperative that the physician should warn a patient of this possibility, particularly if he follows a hazardous occupation.

Relatively common is the association of mitral stenosis with embolism. The prognosis of valvular disease is more unfavorable when more than one valve is involved, and favorable or not according to the tractability of underlying causes, and associated changes in vessels, kidneys, and liver.

This with the implied condition that the patient himself is seconding the physician in his advice as regards mode of life and medication.

**Congenital Valvular Defects.** These consist of alterations in valves, septa, and ducts. In many instances the child dies at birth or shortly after, as often the condition will not permit of life.

These alterations may be due to endocarditis *in utero*, or to congenital defects in development.

From the point of view of the therapist there is not a great deal to be said, as the victims of these conditions rarely live a great while, with the exception of those who suffer from pulmonary valvular conditions, which are likely to allow of longer life.

The heart may be misplaced, either to the right side of the chest, or actually be an abdominal organ.

The auricular or ventricular septum may be incomplete, and as a consequence there may be large gaps between the two cavities.

The foramen ovale may remain patent, with, in some cases, but slight disturbance.

There may be various anomalies of the valves, particularly the pulmonary and aortic, and to a lesser degree at the other sites. Supernumerary and rudimentary valves are sometimes found.

In a case in the author's wards at the Pennsylvania Hospital, the pulmonary artery itself was stenosed. No valves could be found at the pulmonary orifice, but a number of small rudimentary valve formations were found at irregular intervals in the first few centimeters of the pulmonary artery. This patient was an adult.

Pulmonary stenosis is the most common congenital lesion that may call for treatment, and this in no wise differs from that of an acquired condition which, because of heart-failure, calls for appropriate measures.

**Mitral Regurgitation.** This is the commonest of the valvular lesions, and may also be regarded as the least harmful. By this is meant that an ordinarily incompetent mitral valve is less harmful than incompetence of a like degree, say, in the aortic valve. This comparison is more theoretic than real, as it is impossible to measure the degree of incompetence of a valve with any great accuracy, even at autopsy, but the truth of the statement is generally accepted as a result of experience. Mitral insufficiency may exist alone, or may be associated with other valvular lesions, either at the mitral orifice or at other sites.

A certain amount of obstruction may be present, due to thickening of the valves and to deposits in and about the mitral ring.

Furthermore, mitral regurgitation may be slowly replaced by pure obstruction, because of gradual contraction of cicatricial tissue. It is hard to believe that there is any such thing as obstruction without regurgitation, though it is unquestionable that to all intents and purposes marked stenosis is a condition by itself, and any regurgitation which may take place is so inconsiderable as to be ignored.

The characteristic *physical signs* of mitral regurgitation are a soft systolic apical murmur, usually transmitted toward the left axilla, accentuation of the pulmonic second sound, and bilateral ventricular enlargement.

The murmur, usually soft, may be harsh, loud, or quite short or long, and but little significance can be attached to any of these characteristics.

The murmur is not always transmitted, and frequently in very grave cases it is so localized as to be difficult of discovery unless the stethoscope is placed directly at the apex. Sometimes in the case of a heart that quickly changes in size—that is, either dilates or overcomes a dilatation in a few hours—a wrong conclusion that a given murmur has disappeared may be arrived at because of not carefully following the change in the apex position, and thereby failing to hear a murmur heard

but a few hours previously. Mitral murmurs with thrills indicate valvular lesions, and contraindicate mere dilatation from loss of tonicity.

The advent of a mitral murmur, coincident, or nearly so, with cardiac failure indicates a dilatation and functional incapacity of the ring.

It is not necessary here to go further and discuss the symptoms of heart-failure associated with mitral regurgitation, as such symptoms are common to all valvular lesions, and are in reality but those of the muscular failure, the relation of which to valvular disease already has been emphasized.

**Mitral Stenosis.** This condition is the most interesting of all the valvular lesions, and in its proper understanding lies the key to the comprehension of the other derangements. Furthermore, there are several distinct stages of the disease which offer widely different physical signs, and unless the *rationale* of their production be understood, it is difficult to apply treatment intelligently.

Mitral stenosis is, of course, as is well known, more common in women than in men, and is found with great frequency in those who give no previous rheumatic history. It is also quite frequently a progressive lesion, although there are marked exceptions to this where cases go along quite well for many years without great disability, although often with a very rapid and irregular heart, of which they may not be conscious.

On the other hand, after a certain stage is reached, there is no class of cases in which an early and oft repeated breakdown may be more surely prophesied than in advanced mitral stenosis. Hospital cases, in those who are compelled to make their living by manual labor, return again and again, in one to two or three weeks' time, although after a short rest in bed, and the administration of digitalis, compensation is readily established. The social service worker here is a most necessary adjunct to the physician.

Another peculiarity of mitral stenosis is the absence of general dropsy, which in a measure can be explained by the fact that the strain falls largely on the pulmonary circulation rather than on the systemic, as long as the right ventricle holds out.



Cases of mitral stenosis are particularly prone to hemoptysis, and in the absence of a murmur are not infrequently mistaken for tuberculosis and only after a careful elimination of lung disease is the true nature of the case recognized.

The *physical signs* of mitral stenosis vary with the degree of auricular force. Broadbent has, with a great deal of reason, divided the condition into three stages, for all of the symptoms of which he gives appropriate and convincing physiologic reasons.

The first stage is characterized by the presence of a presystolic murmur, followed by a first and second sound of the heart. The murmur is of a peculiar, low crescendo type, and comes right up to and ends abruptly with the first sound of the heart. A presystolic thrill is also present, usually felt just inside and above the apex beat.

The murmur is deep in tone, and vibratory, and indeed, when one is well acquainted with heart conditions, no effort at timing of the murmur is needed, as its *quality* is pathognomonic.

It is perhaps easier also to time by the tactile sense than by the auditory, and the thrill can be most easily recognized as preceding the apex beat.

There is no hypertrophy of the ventricle—or but little—at this or at any other time, except in those cases where there is mitral regurgitation, or a lesion at the aortic orifice.

There is, however, hypertrophy of the right ventricle and dilatation of the left auricle.

In this stage there should be little or no difficulty in making a diagnosis, and also, while the conditions remain as described, the patient will suffer no discomfort or danger.

The second stage, according to Broadbent, is characterized by the disappearance of the second sound at the apex, and by an altered character of the first sound, which loses its normal muscular tone and becomes short, sharp, and loud, and more like a second sound than a first. The apex beat is of a quality that might be expected—rather abrupt and slapping, and devoid of thrust.

This is almost as characteristic as a murmur or thrill, and in many instances, from palpation of the apex beat alone, this lesion may be recognized.

It is in this stage that mistakes in diagnosis frequently occur. Two sounds are heard which commonly are wrongly interpreted to be first sound with murmur followed by second sound, and a diagnosis of mitral regurgitation is established.

The absence or enfeeblement of the second sound is the cause of the confusion, and the peculiar deep-toned quality of the murmur, which should give the clue, misleads the observer into the belief that he is listening to a first sound.

The third stage is characterized by the disappearance of the presystolic murmur, and at this time great irregularity is sure to ensue, although the patient may have no decided systemic disturbance.

The disappearance of the murmur is believed by Broadbent to be due to the giving way of the tricuspid valve, but it is now much more certain that the cause lies in the existence of auricular fibrillation, and the consequent inadequacy of the auricular systole.

There are, of course, many other interesting features of this most interesting cardiac condition, but these fundamental facts are sufficient for a work on treatment alone.

**Aortic Regurgitation.** Aortic regurgitation is quite commonly of syphilitic origin, particularly when occurring alone, and is in the valvular lesion most commonly the cause of sudden death.

It is characterized by a diastolic murmur heard at the aortic cartilage, and often audible with at least equal, or even greater, intensity at the third left cartilage.

The pulse is very characteristic in many cases, and pistol-shot sounds may be heard in the arteries in some cases, particularly in the young, or in those who developed the disease when still young.

The regurgitant murmur is sometimes heard most distinctly at the apex, where also a presystolic murmur described by Flint, is audible. This bruit is due to the impinging on the mitral valves of the blood-currents leaking back through the incompetent aortic leaflets.

A systolic aortic murmur is practically always present when there is aortic regurgitation, though it may not, and usually does not, signify the presence of concomitant stenosis.

Rather is it due to roughness and rigidity of the valves over which the blood-current flows.

Very great hypertrophy is seen in aortic regurgitation, and a patient may have a huge bovine heart for years without being conscious of it, in spite of taking part in games calling for violent exercise.

Angina pectoris is more commonly seen in aortic disease than in disease of other valves, and rupture, at all times a rare condition, more frequently affects the aortic valves than any of the others.

Two observations confirmatory of aortic regurgitation are found in the employment of the sphygmomanometer. Auscultation reveals that the loud systolic "rap" does not disappear at the point expected, but persists as the dial falls to zero. Again, when the patient is prone, the systolic pressure when the cuff is applied to the leg is found to be, perhaps,  $1\frac{1}{4}$  inches (30 mm.) higher than when the observations are made with the cuff on the arm.

**Aortic Stenosis.** The murmur of aortic stenosis is systolic in time, localized at the aortic cartilage, and conducted into the vessels of the neck; it is accompanied by a systolic basic thrill, enfeeblement of the aortic second sound, and moderate hypertrophy of the left ventricle, or no demonstrable enlargement of this chamber whatever. The heart is likely to be slow, and the pulse lingering. The patient is prone to attacks of vertigo and unconsciousness due to fainting.

**Tricuspid regurgitation** is frequently a sequel to heart-failure in association with any of the valvular conditions; it may be congenital, or acquired from rheumatic or other infections.

This murmur is systolic in time, and is heard with greatest intensity over the middle and lower half of the sternum.

The valve is scarcely competent at best, and very readily gives way under back pressure, whereupon symptoms of pulmonary distress appear, and blueness of face and extremities, dropsy, and pulsating liver rapidly follow.

Mackenzie points out that often, in spite of great incompetence of the tricuspid valve, no murmur at all is heard, yet the truth of its existence is proven by the pulsating liver and the ventricular type of liver and jugular pulsations,

**Tricuspid Stenosis.** Tricuspid stenosis is seen in connection with mitral stenosis at times, and usually is not recognized either if alone or if a lesion of the mitral valve also be present.

Broadbent believes that it is to be assumed that tricuspid stenosis has developed in cases of mitral stenosis with great anasarca.

Lesions of the pulmonary orifice or valves are usually congenital. A pulmonary stenosis may be caused by the pressure of an aneurysm.

## ANGINA PECTORIS.

Angina pectoris literally means "pain of the breast," and the term is applied to a symptom-complex consisting of (1) paroxysmal attacks of substernal pain, commonly radiating to and down the left arm; (2) a sense of constriction within the thorax; (3) a feeling of impending dissolution. These symptoms may be present in a given case in greater or less degree, and any one of the three may be totally absent.

There are recognized anginoid conditions to which are given the title of "pseudoangina," which are to be distinguished from true angina. These occur in neurasthenic or hysteric individuals, and should be readily recognized, though sometimes the distinction is not easy. Occasionally true angina pectoris has been mistaken for the attacks of pain common to acute indigestion. If the identity of any particular attack be in doubt, it is better to err on the side of the graver malady, and to treat the patient accordingly, until the correct diagnosis is established.

Angina pectoris is frequently observed among the elderly and in the prematurely aged, due to the existence of arterial change and to alteration of the myocardium. An increasing incidence is noted in the period of mature development, between the ages of 35 and 50, at which time the provocative aortic, coronary or cardiac damage wrought by chronic infections reveals itself.

Angina pectoris is of infinitely more frequent occurrence in males than in females. Statistical studies are most interesting in this connection; Husband's 237 cases give us an incidence

of 60 men to 1 woman; Forbes presents 88 cases showing a frequency of 10 men to 1 woman; Berwenkel's report of 117 cases states the male frequency at 7 to 1; Osler's 40 cases included only 1 woman.

The chief causes of angina pectoris are sclerotic changes in the aorta, coronary arteries, and myocardium incident to the advance of years, and the result of chronic infections. It is probable that the myocardial changes are of at least as great etiologic importance as those in the coronary vessels, as many cases have gone through life without any attacks of angina and yet autopsy has disclosed most severely damaged coronary arteries. A not unlikely view is that the attacks are precipitated by temporary ischemia of the heart-muscle, because of its inability to adjust its circulating power to a sudden demand, such as may follow physical exertion, emotion, or shock.

Next in frequency to the "senile heart" as an etiologic factor, syphilis plays an important rôle. The statistics of Warthin, of Ann Arbor, referred to under Aneurysm in this chapter, indicate the amazing incidence of aortic selection exhibited by the *Spirocheta pallida*. We do not believe that the pernicious activity of the treponema is confined to the aorta alone, and venture the opinion that future researches will reveal its extension to the contiguous coronary arteries and heart-muscle, and establish, statistically, the rôle assumed by syphilis as a frequent factor in angina pectoris. Nor is it too much to expect that further investigations by syphilographers will explain the frequent association of aortic insufficiency with angina pectoris.

Gout, obesity, diabetes, profound mental and emotional disturbances, acute infections, and the toxic effect induced by drugs are classically enumerated as causes of angina. Coffee, tea, and alcohol as probable causes may be ruled out by a two-month abstinence from the suspected indulgence.

The pain, the constriction, and the anguish mentioned in our definition require no further elaboration here, other than the succinct description given by Seneca of his own case: "The attack is very short and like a storm. It usually ends within an hour. To have any other malady is only to be sick; to have this is to be dying."



Dyspnea is rare; the respiratory movements may be shallow, but seldom are they urgent. The pulse is not often altered from its pre-existing condition. Systolic elevations of blood-pressure are not at all constant in the clinical picture, and are as frequently absent as present during an attack. The complexion, as a rule, is at first flushed, then pale; profuse perspiration is to be expected in this anguishing malady.

*Physical signs*, apart from those of concomitant conditions, are absent. The characteristic pulse and infiltrated blood-vessels of arteriosclerosis may be observed, as may also a systolic pulsation, due to a dilated aorta in the second interspace, to the right of the sternal border; an increased area of aortic dullness due to the same cause, may be elicited; but these are not physical signs of angina pectoris: they are indicative only of associated conditions.

Guarded, not necessarily fatal, is the prognosis of this affection, the course of which is dependent upon the degree of exhaustion of the heart-muscle. In young subjects, or in those acute attacks that are induced by toxic agents apparently complete recovery may ensue. Sclerotic hearts with fair cardiac reserve force may live through many years of successive attacks; while a weakened myocardium, with no demonstrable lesion, may succumb early after the establishment of the malady. These incidents are to be well considered in arriving at an estimate of life expectancy, which must be based upon the cardiac reserve force, and upon the opportunities afforded for its conservation.

### TREATMENT.

More latitude in the questions of rest and exercise may be permitted in angina pectoris than in any other circulatory malady. The exhaustion attendant upon a severe attack of "stenocardia" may, of course, necessitate convalescence in bed; attacks induced by exposure to cold may demand that the patient be confined to his room; in advanced myocardial degeneration, the conservation of every ounce of energy may demand absolute curtailment of physical activities. The physician must be guided in his opinion in these matters solely by a careful consideration of each individual case, remembering that, as a general rule, confinement is better borne

by the young, but often results in undesirable conditions in the aged. Exercise is limited to a slow and steady gait on level surfaces, avoiding cardiac strain. Emotional stress, anxieties, mental concentration, and depression of mind are to be avoided. Diet is to be regulated; and, in so far as is possible, the indigestible foods that may induce intestinal disorders or gastric derangements are to be curtailed. Especially is this precaution necessary during an enforced period of rest.

The underlying causes that sometimes may be found provocative of angina pectoris are to be appropriately treated, such as gout, intestinal autointoxication, or dietary indiscretions. Syphilis should be sought for in the middle-aged stenocardiac by the employment of the Wassermann serologic reaction; and, if a strong suspicion be not then confirmed, the diagnostic value of the colloidal-gold test should not be overlooked. If lues be established, the indication for antisyphilitic treatment is plain.

Between attacks, prolonged courses of sodium iodid (less depressant than the potassium salt), in doses of 5 to 60 grains (0.30 to 4.0 Gm.) *t. i. d.* may be used with benefit. Atropin sulphate in  $\frac{1}{250}$ - to  $\frac{1}{40}$ -grain (0.0002 to 0.0015 Gm.) doses per day, continued over long periods of time has many firm adherents, who testify to its efficacy. In those cases which are accompanied by high systolic pressure, the high-frequency current, referred to under Arteriosclerosis in this chapter, is recommended by one no less eminent than Sir Clifford Albutt.

Digitalis has no place in the treatment of angina pectoris. Even when it would seem to be indicated by intercurrent cardiac conditions, its employment should be a matter of much debate. During a paroxysm, pearls of amyl nitrate, each holding 3 minims (0.15 Gm.) crushed and inhaled, often give a prompt and gratifying relief; they may as often disappoint, but are well worth exhibition in every patient. Morphin in  $\frac{1}{4}$ -grain (0.0165 Gm.) hypodermic doses may be required. Liquor trinitrini, 1 per cent. solution, given in 10-minim (0.62 Gm.) doses, may be efficacious where amyl nitrate inhalations fail; this drug, however, requires a lapse of perhaps ten minutes before its therapeutic action becomes manifest. Chloroform should not be used to abate anginal paroxysms; its employment is perilous to the heart.

One further injunction, perhaps not as superficial as it would at first seem, is to be issued against meddlesome interference with the position assumed by the patient during a paroxysm. The sufferer assumes that attitude and position which at the moment causes him the least anguish; to permit an oversolicitous relative or friend to force him into a position that adds to his distress is mistaken kindness, and may do actual harm.

### ANEURYSM.

An aneurysm is a circumscribed dilatation of a blood-vessel. The saccular variety is recognized clinically far more often than is the fusiform type, being more prone to reveal its presence in the thorax by causing erosions of the chest-wall; when arising in the abdominal aorta (a circumstance of 10 per cent. less frequent occurrence), it presents certain physical signs which render its detection through the less rigid abdominal wall a much less complex question of diagnosis. Recognizing that aneurysms may, of course, arise in any blood-vessel, and that they may be of many shapes, and of infinite variety, we shall concern ourselves with the symptoms and treatment of only the more frequent thoracic and abdominal-aortic types.

A primary inflammation and consequent weakening of the vessel-wall, usually at a point where the whirling blood-stream from the heart impinges upon the vessel, and thus further threatens, by erosion, the integrity of the tube, satisfactorily explains the frequency of aneurysm of the ascending and transverse arch or the thoracic aorta. Heretofore it has been customary to ascribe the initial inflammation to toxic substances, syphilis, rheumatism, alcohol, injuries, and even to the traumatism produced by elongated and roughened aortic cusps striking the intima of the aorta, but it appears from recent investigations that syphilis is the cause of aneurysm in all but a negligible number of cases. The patient and laborious researches of Warthin, of Ann Arbor,<sup>13</sup> who, after months of exhaustive examination, was able to demonstrate the *Spirocheta pallida* in the aorta of practically 80 per cent. of the successive, unselected, and routine autopsies which he performed, would seem to indicate that the spirochete exhibits

a marked predilection for the aorta. With syphilis thus so strikingly demonstrated as furnishing the initial damage to the aorta, it requires but little imagination to conceive of the blood-pressure being sufficiently raised by subsequent infections, alcoholism or toxins, to cause erosion of the vessel to the point of aneurysmal formation.

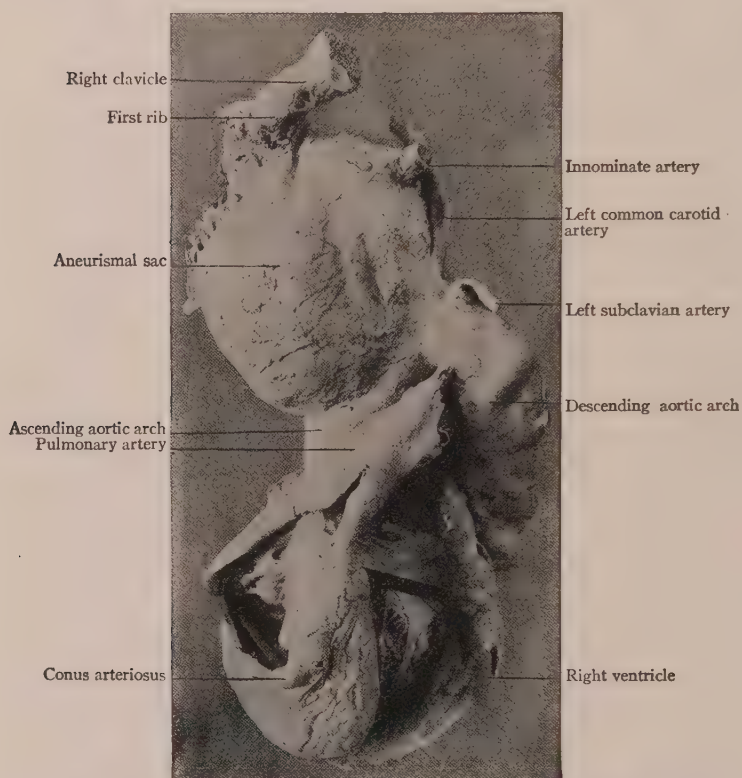


Fig. 16.—Saccular aneurysm of the aortic arch (Philadelphia General Hospital). (From Da Costa's Physical Diagnosis. Copyright, W. B. Saunders Co.)

Aneurysm is usually met with during early middle life. It is six times more common in men than in women. Occupation does not predispose to its occurrence, although violent exertion or long-continued strain may precipitate urgent symptoms in a hitherto unsuspected case.

It should be borne in mind that the symptomatology of aneurysm depends upon the size of the tumor, and upon its location. It may be so small and so deep-seated as to escape detection, and may so dispose itself as not to cause suggestive pressure symptoms, so that its existence is undiscovered in routine physical examinations.

**Thoracic Aneurysm.** Pain is sharp and acute if a nerve be pressed upon, boring in character if a bone is implicated; parox-



Fig. 17.—Aneurysm of the ascending aortic arch (Jefferson Hospital). (From Da Costa's Physical Diagnosis. Copyright, W. B. Saunders Co.)

ysmal attacks may simulate angina pectoris. Dyspnea on exertion may be manifest; brassy cough, with alteration of the voice, due to pressure upon the recurrent laryngeal nerve; contracted or dilated pupils if the sympathetic nerves be pressed upon, together with unilateral perspiration, are symptoms frequently presented. When inspecting the surface of the chest, the patient should be prone, and the eyes of the examiner should be on a level with the sternum; under these circumstances an abnormally situated pulsation, laterally expansile in character, and giving rise to a systolic or diastolic



thrill, may be detected. The back should be carefully examined to discover an aneurysm which may point in that direction. A delayed or feeble pulse is frequently found when one radial is compared with the other; variations in the carotids also may be present; both these phenomena are of significance in locating the position of the aneurysm. Downward tracheal tugging, first described by Oliver, is a valuable, though not pathognomonic, sign of aneurysm. It is caused by pulsations in the arch as it passes over the primary left bronchus, and is elicited by having the patient throw his head back, thus stretching the trachea. The physician, who is standing behind the patient, now gently places his thumb and forefinger under the lower border of the cricoid cartilage, and the tracheal tug is elicited. It is not to be confused with inspiratory movements, nor with pulsations in the neck.

Hypertrophy and dilatation of the heart, long considered classic signs of aneurysm, are found in less than 1 per cent. of cases, according to Howard's autopsy statistics.<sup>14</sup> The heart may, of course, be pressed downward and to the left to accommodate the new growth within the chest. An increase in the area of aortic dullness is the most constant physical sign in early thoracic aneurysm. Rarely the lungs are pressed upon, giving us pulmonary physical signs; or a bronchus may be occluded, and cause an atelectatic lung.

**Abdominal Aneurysm.** Aneurysm occurring in the abdominal aorta, if of appreciable size presents the symptoms of a pulsating tumor, usually to the left of the vertebral column, and above the umbilicus. A systolic thrill and systolic murmur, and even at times a diastolic murmur, may be heard, associated with pressure symptoms. Should the aneurysm point backward, compressing the solar plexus, pain of a lancinating character may be expected; if pressure be exerted on the lumbar nerves, pain will be referred to the region supplied by the nerve, usually in the left groin and affecting the left leg. Gastrointestinal symptoms arise if the growth is directed anteriorly. Erosion of the spine may occur in abdominal aneurysm. Although rare, such an incident was observed in the wards of the Pennsylvania Hospital: a patient admitted for abdominal pain suddenly died; in lifting the body to the autopsy table the spine broke in twain, and a large, sacculated

aneurysm was found to have eroded the spine to the breaking-point. Abdominal aneurysm may be differentiated from other growths in that location by the expansile, pulsating nature of the tumor, and by a maneuver suggested by Osler, who noted that when the patient is put in the knee-chest position, other abdominal tumors change their location, but an aneurysm remains constant in its position and characteristics.

The *x*-ray plate, or preferably fluoroscopy, is our most dependable means of early diagnosis, confirming the opinions established by physical signs and detecting pulsating tumors in cases where a reasonable doubt exists.

The prognosis is distinctly unfavorable, although patients who are well cared for and carefully watched may lead a life of invalidism for years. Relatives should be acquainted with the probability of sudden rupture of the sac, and of its fatal consequences. Recent statistics indicate that the average length of life is two years after an aneurysm has been diagnosed.

#### TREATMENT.

Manifestly, no treatment will restore the lost integrity of the vessel-wall. To the amelioration of symptoms and to the prolongation of life we must direct our attention. If the Wassermann serologic reaction be positive, salvarsan or neo-salvarsan is indicated, in the hope of minimizing the active inflammation of the aorta. The careful physician will not be content with a negative Wassermann, but will, in such a circumstance, employ the colloidal-gold test in an examination of the spinal fluid.

Salvarsan, or "606," is an arsenical preparation introduced by Ehrlich, and is a yellow, crystalline powder, containing about one-third by weight of arsenic. The average dose for an adult is about 0.5 Gm. (7.7 grains) for each 60 kilos (132 lbs.) of body weight. It may be found advisable to administer a smaller initial dose to observe the reaction thereto. Many of the violent reactions have been found to be due to the use of improperly prepared solutions. The ampoule containing the powder is cleansed with alcohol, and the neck filed and broken. The powder is dissolved in a small beaker containing 50 mls (1½ oz.) of absolutely fresh distilled water; the solution may

be gently stirred or shaken to favor solution of the drug. The clear, yellow, acid solution is then neutralized by the addition, drop by drop, of a fresh 15 per cent. solution of sodium hydroxid. The resulting precipitate becomes dissolved as the reaction of the solution becomes slightly alkaline. The solution is then diluted with from 100 to 200 mls (3.3 to 7 oz.) of saline solution, freshly prepared, and is then filtered through sterile cotton. A special needle may be used to insert into the vein through the skin, or the vein may be exposed as in venesection. The median basilic vein is the location of choice for the injection. The patient should receive the drug in the recumbent position, and should stay quietly in bed for several hours afterward.

Neosalvarsan is administered in the same manner. No neutralization is necessary with it. Both these drugs may be given intravenously by syringe, the drug being dissolved in 30 mls (1 oz.) of water. In this manner the vomiting, nausea, and chills caused by larger quantities of the solution may be avoided. The reactions are due, as a rule, to the use of "old" or not properly distilled water. The drug dissolved in oil may be administered by intramuscular injections, but these are, as a rule, so painful that this method is not much used.

The repetition of the dose will depend upon clinical findings, and upon the condition of the blood as revealed by the Wassermann reaction. As a rule, the late secondary or early tertiary conditions, as manifested in early cardiovascular conditions, will be found to present a positive Wassermann test over a long period of time under salvarsan therapy alone, and it must be supplemented by the judicious administration of mercury and the iodids. (For further details of the technic of administering these arsenical preparations the reader is referred to Syphilis, vol. i, p. 80.)

Rest is a prime requisite in reducing arterial pressure, as detailed under Arteriosclerosis (*q. v.*). The patient will assume that posture which is the most comfortable for him, and rarely is it necessary for the physician to interfere in the matter of posture. It is customary for certain physicians to use *veratrum viride* in the routine treatment of aneurysm, but we cannot subscribe to its employment.

Venesection, with the withdrawal of 20 to 30 ounces (591 to 887 mls) of blood, may be of value in relieving pain, and the effect at times is surprisingly long-continued. The effect of an ice-bag applied to the precordium is often gratifying in quieting an overactive heart and in lessening pain. It may remain in place fifteen, or twenty minutes, the interval of application being lengthened according to the comfort of the patient.

The pain of aneurysm is to be relieved by the employment of the less depressant analgesics of the coal-tar derivatives, such as phenacetin or aspirin, in doses of from 5 to 10 grains (0.33 to 0.66 Gm.) at three-hour intervals until effective; if no response is secured, morphin, in the dose of  $\frac{1}{8}$  to  $\frac{1}{4}$  grain (0.0082 to 0.0165 Gm.) is administered hypodermically. The possible induction of the opium habit, as well as a pernicious alteration of the bodily secretions, in a diseased condition which may extend over a period of years, is to be borne in mind by the attending physician. The distressing cough so common in aneurysm as a result of pressure symptoms is difficult of control. Dionin, given in a simple elixir in  $\frac{1}{8}$ - to  $\frac{1}{4}$ -grain (0.0082 to 0.0165 Gm.) doses at four-hour intervals, may be tried. Counterirritation applied to the pectoral region is also of some service.

Iodid of potassium, in 5- to 10- grain (0.33 to 0.66 Gm.) doses *t. i. d.*, is widely employed in the treatment of aneurysm.

Tufnell suggested a treatment for aneurysm, to which Bal-four added the routine administration of potassium iodid, which consists of mental and physical rest, with moderate diet, the patient being confined to bed for a period of from six weeks to three months. The *rationale* of the method is to diminish blood-pressure and to increase the proportion to fibrin in the blood, thus promoting coagulation. For breakfast and supper Tufnell allows 2 ounces (60 Gms.) of bread and butter, 2 ounces (60 mls) of milk; for dinner, 2 or 3 ounces (60 or 90 Gms.) of meat, and 3 or 4 ounces (90 or 120 mls) of milk or claret are permitted. While to many patients this treatment would seem more of an imposition than a therapeutic measure, beneficial results have been reported from its use.

When circulatory failure ensues in aneurysm, and the heart requires support, digitalis is exhibited in moderate dosage, 5 to

10 drops (0.31 to 0.62 mil) of the tincture being given at four-hour intervals until improvement results. As stated under Arteriosclerosis, the effects of digitalis are to be carefully watched for the occurrence of coupled beats or other evidences of untoward effect.

*The Wiring of an Aneurysm.* A sacculated thoracic aneurysm may be treated by the introduction of a fine platinum-gold wire into the sac. It is an operation not to be undertaken lightly, nor by those who have had no experience with the technic employed. The introduction of the cannula through which the wire is passed may result in sudden rupture of the aneurysm—a possibility that should be explained to the patient and his relatives beforehand. The operation has been attended with brilliant results in many instances. One patient whom we have in mind, where the aneurysm protruded from the thoracic wall, and presented a gangrenous area which threatened early rupture, was given a prospective life tenure of six months by a competent surgical consultant. The operation of wiring was performed, and the patient led a moderately active life, in which none of his pleasures were curtailed, for nine years. At autopsy the wiring was found to have formed a mattress of fibrin between the currents of blood and the chest-wall. Wiring should not be performed as a last resort; when it is done moderately early in the condition, it offers more hope of a gratifying result. The possibility that the wiring of an aneurysm at one point may so deflect the blood-stream as to cause a sacculatation to appear at another and inaccessible point in the vessel-wall, should not stay our hand if the procedure is indicated and the case a suitable one.

In the Corradi method, after sterilization of the skin over the aneurysm, fine platinum-gold wire is introduced through a small porcelain- or lacquer- covered cannula into the sac. From ten to fifteen feet (300 to 450 cm.) of wire are introduced, depending upon the size of the sac, and as much as forty-five feet (1350 cm.) have been required in some instances. The end of the platinum-gold wire is now connected to the positive-pole electrode of a galvanic battery, and the current completed by placing a large wet electrode, connected with the negative pole, upon the patient's back. The current is turned on to 5 milliampères, and increased that much every



five minutes, until 50 milliampères are being used. The acid reaction produced by electrolysis about the gold wire produces a firm clot, and by the end of half an hour pulsation in the sac will be found to be notably diminished. No other alloy than platinum should be employed; a copper alloy will be dissolved under the electric current. If an excess of platinum be in the wire, it may be so "springy" as to push aside any fibrin already deposited on the vessel-wall, and, by its resistance, actually push out the walls of the sac and thus defeat the purposes of the operation. At the end of from thirty minutes to an hour, the electrodes are disconnected, and the free end of the wire pushed beneath the skin, the cannula withdrawn, and the puncture sealed. This procedure has been successful in closing the sac in several instances, one of the most beneficial results having been the marked relief from pain, which often occurs within five minutes following the operation. After wiring an aneurysm, the patient should remain perfectly quiet in bed for a period of two or three weeks, to favor consolidation of the clot.

### ARTERIOSCLEROSIS.

Arteriosclerosis is a term used to describe a progressive degenerative change in the intima of blood-vessels, resulting in an inflammatory or a calcareous thickening of the vessel walls. The symptomatology is variable, depending upon the nutritive changes induced in the organ or organs whose arterial supply is thus diminished.

It is convenient to group the causes of arteriosclerosis under three heads, viz: (1) those with an antecedent history of infectious processes, usually long continued; (2) those due to toxic conditions; (3) and, finally, those physical changes incident to the advance of years, especially in the temperamentally high-strung, emotional victims of modern high-pressure living. All three conditions have a similar effect upon the musculature of the arteries in producing an increased blood-pressure, which eventually damages the vessel wall, and in this thickened musculature the subsequent degenerative change occurs.

Among the infections we recognize as etiologic factors rheumatic fever, syphilis, tuberculosis, typhoid fever, and long

continued absorption from suppurative foci, to which the unfortunate term "rheumatism" has hitherto been applied, but to which we now refer under the suggestive terminology "Streptococcosis."

Toxic factors embrace the poisons of alcoholism, gout, plumbism, uremia, and the altered chemistry of the body attendant upon diabetes.

The capillaries are usually the seat of a sclerotic change, which occludes their lumen and thus destroys the circulatory balance between the arterial and venous systems. The arterioles and the arteries become progressively thickened, and impose excess labor upon the heart. The lesions of arterial thickening, calcareous deposits, and calcification of the vessels are not always universally and equally distributed. Changes may be found only in the vessels supplying the brain, or perhaps implicate solely those of the kidneys, liver, or digestive organs.

The frequent association of arteriosclerosis and glomerulonephritis has given rise to much academic discussion as to which of the lesions is cause and which is effect. It may be logically assumed that any congestion or inflammation of the kidney which interferes with its circulation will require an increased effort on the part of the blood-vessels supplying the organ, this overaction eventually resulting in a degenerative change in the vessels.

Owing to the cardiac effort necessary to maintain the circulation, sclerotic areas in the musculature of the heart and hypertrophy of the left ventricle are commonly observed at autopsy. These changes give rise during life to pulse irregularities and cardiac symptoms, which often furnish the first clue to the detection of arteriosclerosis.

Universally regarded as a condition occurring in the later period of life, arteriosclerosis is not confined to the aged, and it may be recognized in middle life. It is of frequent occurrence among males, owing to the more vigorous, rigorous, and exposed mode of life in the man.

The symptoms vary with the organ or organs predominantly affected. If the vessels of the brain are sclerosed, mental fatigue, drowsiness, loss of memory, confusion, and syncope attacks may provoke the rupture of a vessel with its

consequent symptoms of apoplexy. Should the abdominal viscera be partly robbed of their nourishment through arterial degeneration, gastrointestinal symptoms dominate the picture. When the vessels of an extremity are sclerotic, attention is attracted by thermal and sensory changes in the part affected, often with limitations of normal muscular movement and early muscular exhaustion produced by moderate effort. If the kidneys are the organs dominantly affected, the usual clinical evidences of lack of elimination, absorption of toxins, and genito-urinary syndromes are obvious. It should be borne in mind that in any of the conditions mentioned, evidences of beginning cardiac disturbance are quite constantly present in the symptom-complex. Sclerosis of the radials, temporals, or other palpable arteries may or may not be pres-

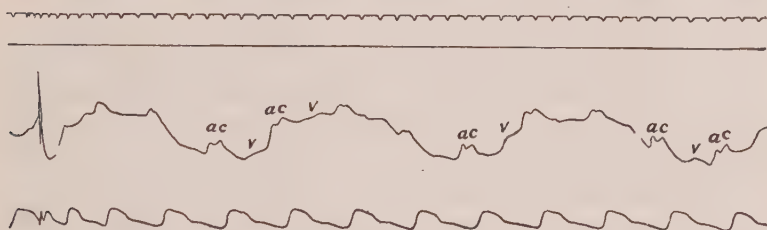


Fig. 18.—Arteriosclerosis.

F., aged 80. Normal rhythm; rate 80; *a-c* interval 0.2 second; loss of arterial elasticity shown by blending of percussion and tidal waves, sustained summit and oblique decline of pulse wave. The aortic notch is clearly shown, but the following dicrotic wave is poorly marked. (Courtesy of Dr. Ross V. Patterson.)

ent; the skin may or may not be dry, cool, relaxed, and wrinkled. We are concerned now with the *early* recognition of arteriosclerotic change in those under our care, and two symptoms in particular should excite the suspicion of beginning arteriosclerosis: the first is high arterial tension, and the second is the evidence presented by a heart working under load.

The earliest symptom of beginning arteriosclerosis may be a curtailment of the normal amount of effort of which the heart has hitherto been unconscious. In a patient, usually past the meridian of life, who complains of breathlessness on moderate exertion, dizziness, confusion, precordial pain or distress, palpitation, cold extremities, insomnia, anginal symptoms, bronchitis, puffiness of the extremities, and dropsy, we

should at once suspect arterial degeneration. If the systolic pressure be constantly from 30 to 60 mm. (0.9 to 1.19 in.) higher than the average pressure for a given age, and is otherwise unaccounted for, arteriosclerosis is probable. The average systolic pressure may be conveniently estimated at 100 plus the age, allowing normal variations therefrom of 17 degrees in either direction, the estimate for women being 10 mm. lower—0.3937 in. If, added to this, a sharp, accentuated first sound at the apex and an intensified second sound at the base are audible, showing increased effort of the myocardium, our suspicions become still more tenable; and if, in palpating the radial, brachial, or temporal arteries, we find an increased sense of resistance in a vessel that remains full between beats, we may consider that the diagnosis of arteriosclerosis may be provisionally made.

Attention is frequently drawn to the heart by a change of its rhythm, due to a premature contraction or dropped beat. If the premature systole is unaccompanied by other signs of cardiac disturbance, it may be disregarded; a dropped beat, however, should keep us on the *qui vive* for heart-block. The sphygmograph or electrocardiograph may be of great value in determining and differentiating these conditions, as well as in keeping us informed of the progress of the affection.

Interest has recently been revived in the comparative intensity of the first and second heart-sounds as a method of determining the functional capacity of the all-essential heart-muscle. It consists of auscultating first over the cardiac impulse at the apex with a stethoscope designed gradually to reduce the sound to the point of disappearance, which point is expressed in figures on a measured scale of the instrument. The second sound is now auscultated at the aortic area in a similar manner; the ratio should be 2:1 in normal hearts. If, however, the first sound be of equal intensity with the second, or if it be less than the second, the indications for absolute rest in bed, to relieve the affected myocardium, are imperative. Electrocardiography has many staunch supporters, who believe it will prove to be the court of last resort in estimating the efficiency of the cardiac muscle and the extent of damage thereto.

The later and classical symptoms of arteriosclerosis are varying degrees of arterial hardening, which include induration, calcareous deposits ("beading," detected by running the finger along the artery), the tortuous and infiltrated radial, the "pipe-stem" artery, and the "snappy" brachial. Added to these are left ventricular hypertrophy, increase of the transverse area of cardiac dullness (normally  $9\frac{1}{2}$  to 11 cm. [ $3\frac{3}{4}$  to  $4\frac{3}{4}$  in.]), frequently a fall of arterial pressure, systolic aortic murmurs, angina pectoris, an ungoverned heart, gangrenous areas due to obliterative endarteritis, pulmonary edema, venous stasis, and chronic invalidism. Such late cases may present either auricular fibrillation or heart-block (*q. v.*), depending upon which part of the heart is damaged, and toward the end of life may develop a pulsus alternans.

Arteriosclerosis is essentially progressive in nature. With its early recognition and the institution of a proper mode of life, we may hope to avoid sudden deaths in unrecognized cases, and may express a guarded but hopeful prognosis to those who present no serious lesion of the all-essential heart-muscle. Indeed, an additional tenure of from ten to twenty years has been obtained by those who were thoughtful in carrying out instructions, and provident in the expenditure of their energy. Far advanced cases are, of course, regarded as critical.

#### TREATMENT.

Arterial thickening, degeneration, or calcification, when once established, is amenable to no treatment. Much value, however, can be obtained from a proper hygienic and dietetic regimen. A rational method of living, the avoidance of physical strain and emotional stress, with strict attention to an easily-assimilable diet, and to the alimentary canal will keep the patient comfortable, and perhaps unaware of the progress of the condition.

High blood-pressure in arteriosclerosis may be safely regarded as an expression on the part of nature to maintain the circulatory balance necessary for the nourishment of the body. If this view be tenable, it appears obvious that the employment of arterial sedatives, such as *veratrum viride*, may well be referred to as meddlesome therapeutics. The daily demand upon the heart is diminished by putting the patient at rest in



bed, interdicting emotional disturbances, securing elimination, and prohibiting the intake of meats and animal broths, thus reducing the protein constituents of food. When such a routine is instituted, the blood-pressure often shows a gratifying reduction, and relieves one of the responsibility of interfering by drugs with a phenomenon not as yet thoroughly understood.

In arteriosclerosis, as in all disease conditions, rest and elimination relieve the burden of wearied nature, and strengthen the patient. Rest is secured by abjuring all business cares and worries and by the avoidance of physical exertion; a few weeks in bed at home or at a sanatorium, an ocean voyage, or change of climate, with its enforced rest and varied mental occupation, may be indicated. Following the initial recuperative period, judicious exercise should be prescribed.

The milder saline laxatives secure elimination by the bowels; the simpler diuretics aid elimination by the kidneys; tepid baths assist in elimination by the skin. Sudden temperature changes or either extreme in hot or cold baths are often dangerous to arteriosclerotics.

If a positive Wassermann reaction demonstrates the presence of the living *Spirocheta pallida* in the body, salvarsan or neosalvarsan are to be resorted to until negative reactions are secured (*v.s.*).

Among drugs, the alterative iodid of potassium has long held first place in the treatment of this condition, its reputation being based on its power to relieve the minor symptoms of dizziness, headache, breathlessness, and exhaustion. It is given in doses of 5 to 10 grains (0.33 to 0.66 Gm.), well diluted, after meals; occasionally it is rapidly pushed to the point of physiologic tolerance, and there maintained at moderate dosage. *Nux vomica* (dose of tincture, 10 to 30 gtt. (0.62 to 1.85 mls) *t. i. d., p. c.*) or its alkaloids may be required for a tonic effect upon the gastro-intestinal tract or upon the system in general.

Insomnia is combated by those hypnotics which often seem peculiarly effective in heart conditions, *vis.*, veronal, tetronal, trional, sulphonal, or medinal, given before retiring, in 5- to 10-grain (0.33 to 0.66 Gm.) doses dissolved in half a cup of hot water. It may be necessary to substitute one preparation for another, inasmuch as the efficiency of hypnotics tends to de-

crease with their accustomed use. Rarely, chloral hydrate, in doses of 5 to 10 grains (0.33 to 0.66 Gm.) is indicated, but this drug is to be cautiously employed. The bromids, in 15- to 20-grain (0.99 to 1.22 Gm.) doses, are effective nerve sedatives. Opium or its derivatives are rarely used in the presence of pulmonary congestion of any degree, for fear of adding the additional weight of retained secretions to the already burdened heart.

The sovereign heart-remedy, digitalis, one of the most effective and most abused drugs in the pharmacopeia, is employed only when circulatory failure ensues, or when degenerative changes in the heart-muscle makes its use imperative, but, even in the presence of these classical indications, we must determine that heart-block is neither impending nor present, for in heart-block digitalis frequently induces alarming symptoms, and perhaps fatal results.

On the other hand, in auricular fibrillation arising in the course of arteriosclerosis, digitalis has a markedly beneficial effect, through its stimulating action upon the inhibitory fibers of the pneumogastric, which controls the area of impulse-formation, the sinoauricular node. The dose of the tincture here is as much as a dram (3.7 mils) a day, reduced as symptoms of compensatory failure disappear. When administering digitalis or digalen, over a period of days, the appearance of a coupled pulse, called "digitalis coupling," indicates the prompt withdrawal of the drug. It is to be remembered that auricular fibrillation may be of sudden onset, and in a greatly damaged heart, attains an alarming degree that threatens life; in such an actual emergency the intravenous injection of strophanthin is indicated, the heroic dosage of  $\frac{1}{50}$  of a grain (0.00132 Gm.) being appropriate.

Heart-block may also give rise to an emergency, in which event atropin sulphate is used, hypodermically or perhaps intravenously, in a dose of  $\frac{1}{100}$  to  $\frac{1}{50}$  grain (0.00066 to 0.00132 Gm.). (*Cf.* p. 216, *et seq.*)

## BLOOD-PRESSURE.

Following the introduction, within the last decade of several mechanical devices for estimating blood-pressure, the majority of the larger insurance companies of the United

States have required that these clinical instruments be used by their medical examiners. The result has been a general adoption of sphygmomanometry, rarely with benefit to the applicant, frequently to the loss of otherwise acceptable risks by the insurance companies, and much to the confusion of the subject of sphygmomanometry. The inrush of physicians into a field where they had little opportunity to make the detailed studies required by the newer clinical method has caused the publication of innumerable and hastily drawn conclusions; really competent observers have spent much time in disproving and controverting unfounded assertions, rather than devoting their attention to establishing the promising future of sphygmomanometry upon a scientific basis. From the haze of contradictory literature surrounding the subject certain conclusions can be drawn, which are here presented for the guidance of the general practitioner:

*Instruments.* There are two types of instruments—those which express the blood-pressure in the readings obtained from a column of mercury within a graduated glass tube, and those operated by a spring. The former is considered more accurate; the latter is more convenient for general use, more easily carried, and, if frequently compared with the standard mercury scale, and corrected in conformity thereto is sufficiently accurate for clinical estimations. It would seem that the spring instrument is the one of choice with the majority of the insurance companies who have replied to circular letters on the subject; it is the one we shall consider in this discussion. The term “blood-pressure,” when it occurs in literature, usually refers to the systolic estimate.

The apparatus consists of a silk armlet, not less than five inches (12.7 cm.) wide, which contains within its folds a rubber “compression-cuff.” To one of the tubes supplying this reservoir an atomizer bulb, with provision for the gradual escape of the air within the cuff, is applied, and to the other tube the dial or register is attached. Blood-pressure is usually estimated by binding the silk cuff about the arm, over the biceps, in order to compress the brachial artery; or the femoral artery may be used, but not when the patient is sitting erect, for in this instance it registers several millimeters higher than the brachial. In the prone posture, the measurements taken

at either point approximate each other. An exception to this statement is noted in cases of aortic regurgitation, where the femoral pressure is 30 or more degrees higher than the brachial in the prone position. The blood-pressure is also higher if measured through the clothing; estimates made when the cuff is applied directly to the arm are more accurate.

*Estimation of Blood-pressure.* The two principal factors upon which arterial blood-pressure depends are: (1) the force of the ventricular contraction, and (2) the degree of peripheral resistance. It is with the idea of estimating these factors that we employ the more exact mechanical devices, rather than depend upon palpation of the arteries, which, by comparison, gives results that are at surprising variance with the instrumental records.

By compressing the bulb of the apparatus, we increase the pressure within the cuff to a point a few degrees above that at which the radial pulse disappears; gradually allowing the air to escape, we note the point where the pulse reappears; this is the systolic pressure. Carefully watching the dial, we observe a point from 40 to 50 mm. ( $1\frac{3}{5}$  to 2 inches) below the high systolic reading, where the greater oscillation of the indicator takes place; this is called the diastolic pressure. The pulse-pressure is the difference between the two.

The above method is generally used, and is mentioned here in order to call attention to its inaccuracies, introduced by the personal equation, and to *condemn* it. The auscultatory method of Koratkov is the only method of reading that should be employed, and is accomplished through the simple maneuver of placing the bell of a stethoscope over the bifurcation of the brachial artery, below the compressing cuff. As the bulb is compressed to fill the cuff, we note that point at which we first hear a sound; continuing the inflation, we hear a gradual increase of sound, which as gradually disappears; note the disappearing point. These observations made on the "up-stroke" of the indicator confirm those now to be secured on the "down-stroke." Compress the bulb a little farther, and then begin the gradual *reduction* of the air. Note the point at which the loud sound first appears, which registers the "*systolic*" pressure; the point at which this clear sound becomes muffled is called the "*diastolic*" pressure.

*Importance of Comparative Readings.* For some inexplicable reason, sphygmomanometry is rarely practised in a systematic manner. It never occurs to many physicians, who take the temperature of a patient at each visit, to estimate the blood-pressure as frequently. It is in the *comparative* study of frequent blood-pressure estimates that its value to both patient and physician lies; cursory and perfunctory examinations are worse than useless, in that they mislead. As an example of the value of repeated readings, it has been discovered in the toxemia of pregnancy that a gradual and progressive rise in systolic pressure is of grave significance, and calls for energetic treatment.<sup>15</sup> When we have a similar frequency of observations, reported from a number of diseased conditions by a number of careful physicians, the subject of blood-pressure may be put upon a scientific basis.

*Normal Standards.* A study of the many tables that have been suggested, whereby we may arrive at the average standard of blood-pressure for a given age, seems to establish the estimates of Faught as normal standards. He gives the systolic pressure in a youth of 20 years as being 120; one degree is added for each two years of life; a normal variation of 17 in either direction is permitted; in women, the record is 10 mm. (0.3937 in.) lower than in men.

A normal ratio of systolic, diastolic, and pulse-pressure is believed to exist in health; it is called the "1-2-3" ratio, in which the diastolic pressure is twice the pulse-pressure; the systolic pressure is three times the pulse-pressure.

One should not lose sight of the exclusion value which attaches to a normal blood-pressure reading in a patient. It aids us in excluding from a consideration of the case those diseases in which hypertension is quite constant in the clinical picture.

*Significance of Pressures.* Despite the many ingenious interpretations which have been placed on systolic readings, high blood-pressure is not a disease. It may often be an expression of an attempt to maintain a *physiologic balance* on the part of nature; we cannot emphasize this conviction too strongly. Temporary rises observed in pain, neurasthenia, excitement, or after exercise, bear out this contention; in arteriosclerosis it is nature's method of supplying blood to vital tissues which may be more or less ischemic owing to capillary fibrosis.



*Hypertension* usually exists in toxemia, nephritis, arteriosclerosis, aortic regurgitation, cardiac hypertrophy, aneurysm, meningitis, exophthalmic goiter, and syphilitic aortitis. *Hypotension*, or lowered systolic pressure, is observed in Addison's disease, tuberculosis, shock, hemorrhage, and asthenic pneumonia. In the past, but little significance has been attached to diastolic readings, but the value of this method of estimating the degree of peripheral resistance is now being appreciated, and promises to exceed systolic estimates in adding to the clinical knowledge of the future.

Sphygmomanometry has not proved its right to be considered as an indication of the functional capacity of the myocardium, regardless of the contentions of its ardent advocates, and despite the vague general opinion to that effect. Our hope for a means by which we can clinically determine the functional capacity of the all-essential heart-muscle lies, at present, in the determined strides which electrocardiography is making in this direction, and in the possible detection of a value existing in the ratio of the first sound of the heart as compared with the second.

We have purposely omitted from this article any of the innumerable "formulas" by which a mathematician may arrive at a calculated estimate of cardiac efficiency by blood-pressure figures; none have so far been adduced which can stand the lime-light of clinical investigation; nor is it likely that dependable formulæ can be thus contrived when one considers the many factors in addition to cardiac muscular action which enter into the complex phenomena of blood-pressure.

A word of protest should be added against the practice of telling patients that they "have a high blood-pressure." Remarks similar to this frequently cause much distress, and until such a time as the profession better understands the significance of high blood-pressure, so that we may more intelligently interpret it for our patient, we are in the position of the blind leading the blind.

#### TREATMENT OF HYPERTENSION.

As the reader has already probably gathered from the preceding consideration, the indications for administering vascular sedatives are rare indeed. Rest in bed; freedom from

anxiety, depressing emotions, and excitement; free catharsis by the use of saline purges, given in small doses, and frequently repeated; elimination by the kidneys and by the skin, will do as much to reduce hypertension, and do it far more safely, than will the employment of drugs. Attention will, of course, be given to the diet, which should be carefully selected, and of a limited quantity. Foods that contain a high percentage of protein are interdicted, familiar examples of which are meat, eggs, fish; shellfish, fowl, cheese, peas, and beans. When emergency demands, venesection may be indicated. Drugs are employed only in exceptional instances, their power for good being questioned, and their possibility for harm in other directions being generally admitted.

### THE USE OF CARDIAC DRUGS.

"When called to guide a patient through an illness, the physician should be constantly a watchman, and a therapist only when necessity arises" (Hare). The drugs which have been used in cardiovascular disease are legion; those of proved efficiency are few. Remedies affecting the heart indirectly by their effect upon other organs cannot be considered as cardiac drugs. In this consideration we will concern ourselves with remedies of demonstrated value, leaving for future clinical and graphic researches the final verdict as to whether there shall be included among dependable cardiac remedies those drugs which have been administered empirically in the past.

The newer remedies, such as epinephrin and pituitary extract, are cardiac potentialities, and as such will here receive the mention which they have so far earned. Drugs of time-honored administration, to which present investigation attaches little cardiac effect, receive a brief allusion.

**Digitalis.** This sovereign heart-remedy was first brought to the attention of the profession by Withering, who wrote "An Account of the Foxglove" in 1785. It is interesting to note the astute observations of this pioneer, who, in speaking of the diuretic action of the drug, avers that "Digitalis seldom succeeds in men of great natural strength, of tense fibre, of warm skin, of florid complexion, or

in those with a tight and cordy pulse. If the belly in ascites be tense, hard, and circumscribed, or the limbs in anasarca solid and resisting, we have but little hope. On the contrary, if the pulse be feeble and intermitting, the countenance pale, the lips livid, the skin cold, the swollen belly soft and fluctuating, or the anasarcaous limbs readily pitting upon pressure of the finger, we may expect the diuretic effects to follow in a kindly manner." With what prophetic foresight he thus confirmed the observations of today, when he allowed but little value to the drug in arteriosclerotics with high arterial pressure and associated dropsy, and in admitting a beneficial action in the clinical picture of auricular fibrillation which he has so well painted! Controversial storms must have waged around Withering's head, for in closing his preface he seeks the solace of saying: "After all, in spite of opinion, prejudice, or error, time will fix the real value upon the discovery, and determine whether I have imposed upon myself and others, or contributed to the benefit of science and mankind."

Digitalis is derived from the dried leaves of the perennial *Digitalis purpurea*, or Foxglove, collected from plants of second-year growth as they are about to flower. While many glucosides have been separated by chemists, the drug does not have an "active principle" that is universally admitted. The true therapeutic effect of the remedy is best secured by the employment of a physiologically tested tincture or infusion, which combines *all* of the qualities claimed for several "isolated principles." The employment of "digitalis derivatives" may account for the absence of digitalis *results* occasionally complained of by physicians. This statement is not to be construed as expressing an unfavorable opinion of standardized tinctures or extracts as prepared by reputable houses; it is intended to convey the conviction that alleged "digitalis-active principles" do not give satisfying digitalis results, any more than do the stale tinctures occasionally dispensed. It should be the custom\* of physicians who see many heart cases to select a physiologically tested and standardized tincture of digitalis, as prepared by any one reputable pharmacist, and at the same time to assure himself of the potency of the hypodermic tablet which he proposes to use; by employing these two preparations to the exclusion of others, he soon becomes familiar with

the results to be expected from a given dose, and becomes adept in the skillful employment of his remedy.

When employed in hearts of disturbed mechanism, the physiologic effect of digitalis in therapeutic dosage is shown in (1) decreased atrioventricular conduction; (2) increased force of the ventricular contraction. By decreasing the conductivity of the A-V node, it slows the pulse and increases the length of diastole; by increasing the force of the ventricular contraction, it increases the pulse force and

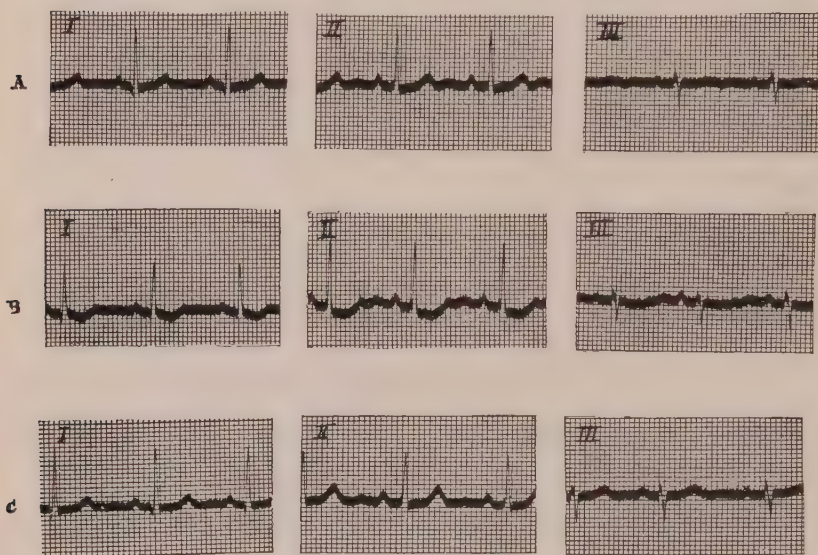


Fig. 19.—Influence of Digitalis on the Electrocardiogram.

The leads in these figures are arranged horizontally, the reverse of the usual order, for ease of comparison. (A) Control curve. Before administering digitalis. (B) After 1.4 Gm. of digitalis had been given. Note (a) diminution in height of T-wave; (b) downward slope from end of R or S to T. (c) This curve, taken nineteen days after the drug was discontinued, is one of a series which shows a gradual return to normal, and is the first of said series to virtually reproduce the control curve A. (Courtesy of Dr. Alfred E. Cohn, of the Rockefeller Institute for Medical Research.)

raises arterial pressure. We are fully aware of the mass of literature relating to the action of the drug on the pneumo-gastric nerve, on the sympathetic fibres, and on the arterioles; we feel that emphasis of the physiologic action, already briefly stated, is quite sufficient for the purposes of the clinician who would administer the drug understandingly.



In the form of the infusion, digitalis is prized as a remedy to relieve dropsical effusions. This it does by removing the congestion of the kidneys and by improving the blood-supply to these organs, rather than by any action on the renal cells. By thus improving the circulation of the kidneys, it is a depletant of accumulated body effusions.

No definite limit can be placed on the amount of digitalis necessary to produce and maintain a desired physiologic result. The old rule of "giving the drug until the pulse becomes regular" has probably been responsible for many deaths by inducing heart-block, and would seem to explain the "cumulative action" and "digitalis deaths" of medical literature. When we recall that the investigations of Cohn<sup>16</sup> demon-

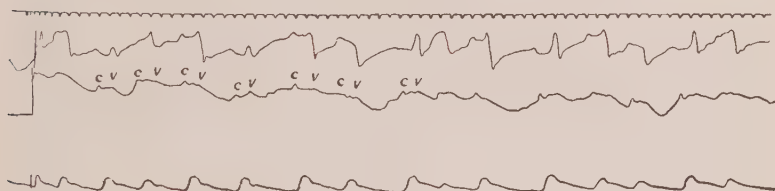


Fig. 20.—Digitalis Coupling in Auricular Fibrillation.

The cardiac rate, previously 150, under full doses of digitalis, has been reduced to slightly above 60. A pulse deficit of 40 has been reduced to *nil*. The tendency to pairing (or tripling) of the pulse-beats is clearly shown. Digitalis should be discontinued. (Courtesy of Dr. Ross V. Patterson.)

strated that in some instances digitalis affected the heart within thirty-six hours of its administration, and that the effect persisted as long as twenty-two days after withdrawal of the drug, we can appreciate how unreasoning abuse of the remedy would induce not only heart-block, but complete exhaustion of the laboring ventricle as well. (See Fig. 19.)

Fairly dependable symptoms of physiologic tolerance are nausea, vomiting, and headache, but unfortunately these symptoms are not reliable guides, as they may ensue after the first dose, may not set in for several days, or may utterly fail to appear. With ordinary physical signs to guide him, the clinician should revise the initial dosage of digitalis (1) upon the appearance of a first sound of good muscular quality at the apex; (2) upon the appearance of a gradual equality in the ventricular and radial rates in cases of auricular fibrilla-



tion—in other words, when the *pulse deficit* is only ten points less than the simultaneously counted rate at the apex—administration of the drug should be carefully guarded; (3) upon the appearance of “digitalis coupling,” often revealed on thoughtful radial palpation, in patients in whom the drug is used. (Fig. 20.)

It is almost trite to remark here that prolonged use of digitalis over periods of months is to be condemned, unless frequent professional observations so dictate. One patient recently seen had been taking digitalis, upon his own initiative, for a year and a half. A *non-repetatur* upon a prescription will save a physician from the censure attached to the medical attendant in this instance.

Digitalis is the remedy *par excellence* in auricular fibrillation, in which condition its chief beneficial effects are observed, and upon which its reputation rests. It is of marked value in auricular flutter. Paroxysmal tachycardia, which may induce exhaustion of the cardiac muscle, may call for the support afforded by this drug, as also may alternation of the heart. Heart-block is a contraindication to digitalis, unless the block be complete, in which latter event it may increase cardiac tone. Hypertrophy and dilatation do not call for its employment, unless circulatory failure ensues.

If the physician will but think of digitalis as a drug to be used *only when especially indicated for definitely recognized conditions*, both laity and profession will be benefited by the thought. The detection of murmurs does not call for digitalis, although the drug may, of course, be indicated in valvular lesions with evidence of exhaustion of the cardiac muscle. No drug can correct a chronic valvular lesion, in the manner that lubricating oil may temporarily correct the leaking valve of a pump.

*Strophanthus*. This drug is derived from the seeds of African plants, *Strophanthus hispidus* and *kombé*. It is similar to digitalis in its cardiac action as clinically observed; experiments now being conducted by graphic methods will give us further light on the subject.

*Strophanthus* is to be borne in mind as the remedy of choice *when digitalis fails*. It is often administered to those patients who have an idiosyncrasy to the latter drug, or in

whom digitalis produces gastro-intestinal symptoms—yet it is not always without this untoward effect itself. If given to a patient who has previously had large courses of digitalis, it should be very cautiously employed.

Tincture of *strophanthus* is given in doses of 5' to 15 minims (0.3 to 1 Gm.) *t. i. d.* The active principle, *strophanthin*, is used hypodermically in a strength varying from  $\frac{1}{150}$  to  $\frac{1}{50}$  grain (0.0004 to 0.0012 Gm.), the last-mentioned dosage being indicated only in urgent cases.

*The Nitrites.* Nitroglycerin, amyl nitrite, and sodium nitrite are dependable circulatory aids where immediate effect is desired; they are, however, fleeting in character, the reaction not being long sustained, and for this reason are administered at three- or four- hour intervals.

The nitrites cause acceleration of the pulse by a reflex action on the vagus center in the medulla. There is no experimental proof that they have any direct action upon the heart-muscle; hence a much-damaged myocardium does not contraindicate their employment when their action is otherwise desired, as in relieving the hypertension of angina pectoris. We should think of the nitrites as *relieving arterial spasm*, for it is in this direction that they exert their most pronounced therapeutic effect. The value of this form of medication in the cardiovascular failure of acute infections, such as lobar pneumonia, is to be also borne in mind.

The dose of the nitrites is as follows: The *spiritus nitratis* is a 1 per cent. aqueous solution of nitroglycerin, and is given in 1- or 2- drop (0.05 or 0.10 mil) doses, sometimes gradually ascended. Tablets of nitroglycerin (*tabellæ trinitrini*) each contain  $\frac{1}{200}$  grain (0.0003 Gm.). The *perles* of amyl nitrite contain 2 minims (0.10 Gm.) each, and are to be crushed in a handkerchief and inhaled for the relief of arterial spasm.

*Salvarsan.* To include the recently proven antisyphilitics under the heading of Cardiac Drugs is an innovation; to exclude them from the classification of heart remedies would be to deny the gradually accumulating evidence which will eventually determine the frequent etiologic relation between syphilis and cardiovascular disease. The preparation and administration of salvarsan and neosalvarsan are described under Aneurysm. (See p. 263.)

*The Iodids.* These are valuable adjuncts in the treatment of circulatory disorders, either through their alterative effect or by virtue of their action in specific disease. Under Arteriosclerosis administration and dose are discussed. (See p. 272.)

*Atropin.* This alkaloid of belladonna is extracted from the roots or leaves of "Deadly Nightshade." Though at first slowing the heart by stimulation of the vagus center in the medulla, this slight initial effect soon passes off, and the heart-rate increases, owing to a paralysis of the terminal inhibitory fibers of the vagus nerve. Atropin probably increases the conductivity of the *A-V* bundle; hence it is the remedy indicated in all degrees of heart-block, the subcutaneous dose of the sulphate being  $\frac{1}{50}$  grain (0.0012 Gm.), to be repeated when the effect has disappeared. The action of the drug on the bundle neutralizes the effect produced by giving digitalis in excess.

Tincture of belladonna is administered in the dose of 5 to 40 minims (0.3 to 2.6 Gm.). The drug produces dryness of the fauces, dilated pupils, sometimes an erythematous rash, and perhaps a talkative delirium, in full medicinal dose—physiologic effects to be discounted by the physician seeking a cardiac response, and yet most uncomfortable symptoms, strenuously objected to by the patient who is to take the remedy for a long period.

*Morphin.* This drug slows the heart-rate, not by a direct action upon the cardiac muscle—for there it has no effect—but by stimulation of the vagal center. Hence, myocardial lesions do not contraindicate the drug, except in so far as its effects upon other parts of the body add to the heart's embarrassment; morphin checks all bodily secretions except those of the skin.

The pain of myocarditis and angina pectoris, the rate in tachycardias, which is enhanced by excitement and restlessness, the intractable insomnia of some varieties of heart disease, and the dyspnea of others, all may call for the employment of morphin.

Morphin sulphate is administered in doses of  $\frac{1}{8}$  to  $\frac{1}{4}$  grain (0.007 to 0.15 Gm.), repeated at two- or three- hour intervals, until the desired result is secured. When indicated at all, morphin is indicated *to effect*. To curtail the full therapeutic value of the drug by an arbitrary limit of dose is an

error of judgment too frequently seen—an error which time may, perhaps, eradicate from the professional mind.

*Epinephrin.* This drug is indicated where there is evidence of acute circulatory collapse, with falling blood-pressure. It is fleeting in action, and should not be used where a sustained circulatory effect is desired. Janeway used a large dose of 4 mils (1 dram) of a 1:1000 solution with most amazing results, in the restoration of a patient apparently moribund.

*Pituitary Extract.* This is similar in action to epinephrin, the difference being in degree. The former drug, while slower in action, and less decided in effect, maintains its circulatory stimulation for a longer period than does epinephrin. Extract from the infundibular portion of the pituitary gland manifests the greatest physiologic effect.

*Caffein.* As a result of their animal experimentations, Pilcher and Sollmann<sup>17</sup> conclude that caffein causes: (a) cardiac stimulation; (b) increase of heart-rate not due to vagus depression; (c) vasodilatation through peripheral depression of the vasoconstrictor mechanism; (d) central vasoconstrictor stimulation to be generally ineffectual. From this we deduce the tenable hypothesis that caffein probably accelerates the heart action by direct stimulation of the heart-muscle. Caffein is administered in dosage of from 2 to 4 grains (0.12 to 0.25 Gm.).

*Drugs of Doubtful Utility.* It may not be altogether wise nor judicious to discard time-honored remedies whose reputation for cardiac efficiency bears the medical testimony of many generations of accustomed usage; yet it is quite appropriate, in this age of scientific investigation, that we require remedies to meet the standards set for them by other remedies, equally as honored by age and by accustomed usage, which show their action in a manner satisfying to a laboratory investigation. If practising physicians will avail themselves of the *proven* drugs, and if the coming generation of medical men will avoid the use of remedies now placed under the ban of cardiologists, then will cardiac therapy advance immeasurably as the future necessity for detailed clinical and graphic study is thus made plain.

*Alcohol* raises for a few moments the systolic pressure, and thus acts as an apparent circulatory stimulant; it cannot, however, be regarded as a *true* circulatory stimulant, inasmuch as it decreases cardiac efficiency, raises disproportionately the

diastolic pressure, and lowers pulse-pressure, according to the exhaustive investigations of Lieb,<sup>18</sup> corroborated by many others. Alcohol is no longer considered a *food*, for it has been determined that its oxidation in the body is a *protective* oxidation (as is that of uric acid, xanthin bodies, leucin, etc.); it is *not* oxidized for the purpose of being used by, or stored up in, the economy as a food.<sup>19</sup>

*Ammonia* reduces heart-rate by reflex nervous inhibition. The effect, when it does appear, rarely lasts longer than a few minutes.

*Camphor* affords no direct evidence whatever that it favorably affects the heart-muscle. Its use in auricular fibrillation, from recent reports, makes it of doubtful value in that condition.

*Strychnin*. Newburgh,<sup>20</sup> in his experiments on patients, found that none were benefited by strychnin; compensation was not improved in the slightest; and he concludes that neither pharmacologic nor clinical evidence justifies its use in the treatment of either acute or chronic heart disease. Pilcher and Sollmann<sup>21</sup> showed that strychnin had no effect upon the heart; that it had no direct action upon the blood-vessels; that it produced no marked effect upon blood-pressure. Despite these observations, it is quite possible that the beneficial results of strychnin administration may be due to the action of the drug in improving *systemic tone*.

### THE NAUHEIM BATHS.

Balneotherapy in the treatment of chronic diseases of the heart, as introduced by Prof. Dr. Theodor Schott, of Bad-Nauheim, has been the center of many storms of professional opinion as to its therapeutic value. That the employment of these baths has proven efficient in many cases of cardiovascular disease is supported by a wealth of clinical observations; any work on the treatment of heart disease that does not give to the subject that consideration to which a multitude of successful results entitles it, would be incomplete. In order that we may present the subject as its distinguished sponsor would have it presented, we give in the following pages a description of the Nauheim baths, as translated by Dr. S. Lewis Ziegler, of Philadelphia.<sup>22</sup>



## METHODS OF BALNEOLOGIC TREATMENT.

"Here, as is often the case in medicine, merely general rules can be formulated. It stands to reason that a careful physical examination of the patient must be made, since only in this way can a strictly personal treatment be outlined; this should never be disregarded, because these baths exert a most decided action in all systemic diseases, and especially in affections of the heart. According to the method of application, favorable results may follow, just as readily as, conversely, unfavorable effects may be produced. It has frequently been emphasized by us that even here a constant control by the physician is essential. The symptoms in heart affections may undergo sudden changes, and especially during balneologic therapy; it is therefore, advisable to examine the heart frequently *before, during, and after* the bath. The methods which have proved most successful in my hands are largely the following:

"It is best, more especially with severe cases, to commence with a simple salt-water bath. Since the water at Nauheim contains between 2 and 3 per cent. of sodium chlorid and as much calcium chlorid per thousand, it may be necessary to dilute this still more. The duration at first should not exceed eight to ten minutes, in severe cases not over five. The temperature should commence at 93° to 95° F. (33.8° to 35° C.), and should be reduced but slightly during the first week. One must be specially careful with anemic and weak patients, and with those who are easily chilled. On the other hand, however, even in patients with weak, rheumatic hearts, one should not exceed a temperature of 95° F. (35° C.), since a tonic action on the heart will not be gained. It is, therefore, preferable to administer cooler baths, and to make them of shorter duration. In the first half to one minute the patient, while remaining quiescent, may experience a feeling of chilliness; then, however, a sensation of full comfort should occur, partly owing to the warming action of the bath on the skin, and partly from habituation. If, however, after a minute's quiescence this does not result, but rather the slight feeling of cold persists, then the bath must be slowly and carefully warmed to a temperature which is just sufficient for

the purpose. In the majority of cases, as the cure advances, cooler and cooler temperatures are tolerated, and may be used with benefit.

"One should avoid, if possible, a second or recurring chill while in the bath. By this we understand that a patient who had become chilled on entering the bath, had later regained his warmth, and shortly thereafter had commenced to feel chilled again, after he had remained quiet for some time. Such a bath was too prolonged in relation to its temperature. The temperature should either be rapidly raised or the patient should leave the bath at once. During the following days warmer baths should be given.

"Many patients, especially those who suffer readily from dyspnea, cannot tolerate complete baths at first. They find the pressure over the cardiac region very troublesome. It is best to recommend that such persons should not be immersed in the water deeper than to the level of the nipples. Gradually they also become accustomed to the full bath. Partial baths may easily produce ill effects, and should, therefore, be avoided. Patients suffering from heart disease should bathe neither on an empty stomach nor on a full one. Most suitable of all is the forenoon, about one to two hours after breakfast, or, if this cannot be arranged, then the late afternoon, from three to four hours after the midday meal.

"If the baths are well tolerated, the stronger concentrations may gradually be employed. First, the concentration of the salts should be increased, especially that of the calcium chlorid. At Nauheim we obtain this by the use of the mother-liquor derived from the spring, which contains 30 to 40 per cent. of calcium chlorid. After these follow the baths containing carbonic acid, in a quiescent state, and later those rich in free carbonic acid gas; for these we employ at Nauheim, in rotation, the Thermal, Thermal effervescing, and Sprudel effervescing baths, as well as the effervescing flowing or Strom-Sprudel of the individual springs, with their different temperatures and their varying concentrations of salts and carbon dioxid.

"Patients with heart disease, without exception, require *days of rest*, on which the bath is suspended; in certain instances, especially with severe cases, a *pause day* is necessary,

even after the first day; usually, however, after the second day. Later, three or four baths may be given on successive days. Simultaneously, an extension in the duration of the bath also takes place; it is, however, seldom advisable, particularly in severe cardiac lesions, to prolong them beyond eighteen or twenty minutes. After each bath the patient should be wrapped in hot towelling, and rubbed down vigorously, so that the skin becomes red and warm. He should then resume his clothing, and immediately seek his room, where he should *rest in bed for at least one hour*, under a suitable covering, in order that the body may be rested and maintain an equable warmth. During this *rest-period* the mind should be kept quiet, and all reading avoided. In the further course we should endeavor to obtain a continuous, but, nevertheless, prudent stimulating action of the baths. The baths should be given always slightly cooler; always for longer periods, and at more frequent intervals. An exact supervision by the physician in regard to this should be constantly exercised. The result of today's bath is the criterion for tomorrow's orders.

"Owing to their strong content in salt and carbonic acid, the Nauheim baths can gradually be taken fairly cold, and can be safely borne by patients with cardiac affections. In this way there is a possibility of hardening such sufferers, little by little, in order to make them resistant toward cold, and particularly toward muscular rheumatism, which is naturally of great importance for heart patients.

"During menstruation the baths should be discontinued, as an abnormally large loss of blood may be caused by such powerful baths, and this must be especially avoided in cases of heart disease.

"The *summer months* are the most suitable for balneotherapy. In mild cases, from *three to six weeks* are sufficient, to which may be added with advantage an *after-cure* in a moderately high mountainous district, not over 1000 to 1200 meters (3000 to 4000 feet). In severe cases, on the other hand, the treatment should extend over several months, and it is then advisable to divide the cure into two parts, and to separate them by a short residence in a mountainous region. To form an exact estimate of the number of baths at the beginning of treat-

ment is not possible, since, apart from the severity of the case, patients react quite differently to the baths. It can be readily understood, therefore, why it is quite out of the question to formulate such a scheme. During the winter many patients with heart disease require residence in a southern climate, in order to remain in the open air to the fullest extent.

"It is now possible for those suffering from heart disease, who are not in a position to take the cure by means of the natural baths, to imitate these baths at home, to a certain extent. My brother and I have laid down exact directions to this effect. One should employ for this purpose preferably the natural Nauheim bath salts, or, if these are not available at the moment, make use of the most important of their saline constituents, namely, *sodium chlorid* and *calcium chlorid*, in the correct proportions—2 per cent. of the former, and 1 part per 1000 of the latter. These quantities may be increased when stronger baths are indicated. The carbonic acid is best obtained from sodium bicarbonate and hydrochloric acid; both are used in the form in which they exist in commerce. The chemical equivalents indicate in what proportion these ingredients are to be added to the bath. With the strong solution of hydrochloric acid (equivalent to 42.8 per cent.) equal quantities of hydrochloric acid and sodium bicarbonate should be employed. With the dilute hydrochloric acid a correspondingly larger quantity of this solution is necessary. The sodium bicarbonate, commencing with 100 grams (3 oz.), and gradually increasing to 500 (15 oz.), 1000 (30 oz.), or even to 1500 grams (45 oz.), as the baths progress, should be dissolved in the bath-water simultaneously with the other salts (sodium chlorid and calcium chlorid), which must also be increased, in proper proportions for these stronger baths. An excess of bicarbonate of sodium is always advisable for the protection of the bathtub. After the temperature of the water has been properly regulated, an amount of hydrochloric acid equivalent to the quantity of sodium bicarbonate already dissolved in the bath is poured directly on the surface of the water from a small-mouthed bottle and distributed well over it. One should avoid any additional agitation of the bath-water, as otherwise the carbon dioxid will readily escape into the air. The layer of carbonic acid gas which forms on the

surface of the water during its preparation should be driven off with a towel before the bath is used, so that the patient will not breathe it. In this way the carbonic acid gas will continue to be evolved for a considerable time, probably a half-hour or more.

"Instead of hydrochloric acid one may employ a milder acid, as, for example, formic, citric, or tartaric acid, since these are less liable to attack the sides of the tub; of course, they must also be added in amounts corresponding to their respective equivalents (about 2 parts of acid to 1 part of sodium bicarbonate).

"By the use of such baths as these, many errors have become apparent. For instance, it is held by some that a plain salt-water bath, or, what is still more common, a plain carbonated bath, is sufficient for the treatment of chronic heart disease. If we desire to obtain an increasing tonic action, so far as may be possible with such artificial baths, then we must even here increase in a systematic manner the dosage of the ingredients, salt, calcium chlorid, sodium bicarbonate, and hydrochloric acid, and adjust the temperature and duration of these baths to the condition of the patient at the time being.

"The employment of the ingredients in definite and fixed doses, as they are prepared and sent out by certain factories (known as "acid cakes"), has led to the evolution of carbon dioxid in improper proportions, and, above all, to the quantity of the gas increasing by leaps and bounds. These are positively dangerous. A considerable number of patients have had, as I have been able to convince myself, imperfect or untoward results from the fact that the baths formed with such prepared doses, owing to their faulty adjustment, were suitable neither for the case as such, nor for the temporary condition of the sufferer.

"It is self-evident that even in the employment of artificial baths a constant supervision by the physician is essential, if satisfactory results are to be obtained. And often a good result is only to be secured when it is practicable to remove the patient from business and family worries into pure air and new surroundings. Suitable nourishment also plays an important part in these cases. If properly used, favorable results can be obtained with artificial Nauheim baths in a



certain proportion of cases. Naturally, the number of cases to be benefited must necessarily be limited by the circumstance that the strongest of these baths—the effervescing and the effervescing flowing baths—cannot be imitated artificially.”

### EXERCISES IN CHRONIC HEART DISEASE.

For those cardiopaths who cannot go abroad to secure the beneficial results which follow the resistance exercises of Schott, or the graduated exercises of Oertel—a method of treatment which has proved most successful in selected cases—we here present a consideration of the subject which will enable the general practitioner to intelligently carry out the principles as employed at Nauheim.<sup>23</sup>

*Methods of Gymnastic Treatment.* By means of regulated gymnastic exercises, effects can be obtained similar to those of balneotherapy. The bath produces its action by way of the sensory nerve-tracts; the gymnastics, as already mentioned, through other nerve-tracts. The essential characteristics of the gymnastics are as follows: The movements employed must always be carried out slowly, and with such degree of power as the momentary condition of the patient will permit. In order to procure this simultaneous retardation and increase in strength, resistance is necessary, which is supplied by a second person, the “gymnast,” or operator; this is the simple passive *resistance gymnastics*. Or, the resistance is produced by the patient himself, through the simultaneous contraction of antagonistic muscles; this form we have called *gymnastic exercises with self-resistance*. Regarding the employment of gymnastics, the following general regulations may be stated:<sup>24</sup>

1. The movements should alternate with one another in such a manner that, according to their arrangement, new groups of muscles are continually being brought into activity. After the movements have taken place over the whole of the skeletal musculature, they can eventually be repeated in several cycles if the patient still feels sufficiently fresh. In this way a one-sided fatigue is most effectively prevented.

2. This general activity of the skeletal muscles can be attained by means of a very simple geometric arrangement of the movements. Usually we employ the following scheme:

(A) Movements of the extended arms in three vertical directions, one after another.

(a) Sagittal, toward the front from the position of downward extension, upward to near the temples, and from there downward again.

(b) Frontal, laterally upward to the temples, and backward.

(c) Horizontal, brought together and apart again in a horizontal direction.

(d) The fully extended arms are rotated on their axes outward and inward to the farthest extent, which includes pronation and supination.

(B) While with the free articulation of the shoulder-joint a selection of the directions of movements was necessary, the movements for the elbow-joint and wrist are determined by nature. Flexion and extension, with radial and ulnar abduction; the rotations have already been carried out under (A).

(C) Movements of the body, bending forward from as far back as the patient can bend, and the reverse; side movements from the extreme left to the extreme right, and *vice versa*, as well as rotation of the vertebral column on its axis, in both directions.

(D) The extended leg should be raised under resistance, straight forward and upward, and again lowered against resistance; then again to the right and to the left, and raised and lowered toward the back.

(E) The directions of movement for the knee- and ankle-joint are also determined by nature.

It is not necessary to carry out all of the movements on each occasion.

3. One can train up men to act as gymnastic operators in a very short time, if they have sufficient conscientiousness and intelligence. Of course, they must previously have gained a certain amount of anatomic and physiologic knowledge. It is of advantage, in many cases, to teach a member of the patient's family, so that the sufferer can always have someone at hand.

The *gymnastic operator* must learn:

(a) *The Application of Resistance.* According to the relation of his strength to that of the patient, he should apply the resistance at a higher or lower position, in the latter case with the advantage of greater leverage in his favor. The resistance should always be applied on the advancing side, as, for example, on the anterior side of the forearm when the two arms are being approached together horizontally, and on the dorsal side when they are being horizontally separated again by the opposite movement. Similarly, on the upper and lower leg he has to exert pressure at one time on the front, at others on the outer, inner, or posterior side of the limb, according to the momentary direction of the movement. The resistances for the bending of the body are applied in front over the manubrium sterni, or at the back over the lumbar vertebræ; those for extension of the back on the neck and on the xiphoid process. To check the rotation of the body, the operator stands at the side, and places the right hand in front of the advancing shoulder, and the left hand behind the retreating shoulder, and so on.

The operator should exert pressure on one side only, and never grasp the limb, because he may thus quite easily, and unconsciously, prevent the movements instead of assisting their accomplishment against resistance.

(b) The operator must, in fact, commence with the intention of moving the limb which is undergoing exercise in a direction opposite to that in which it is being directed, but he must always allow the patient to have the upper hand. From my own experience, I can recommend only these so-called eccentric movements for patients suffering from heart disease.

(c) The resistance must be so calculated that the movement may succeed in a slow and regular manner, but should never be so powerful as to stop the movement completely during its progress, or to allow it to progress only by fits and starts. The hand of the operator should always exert approximately the same even pressure on the patient's limb while it follows the limb through the movement.

4. While the previous regulations for the use of curative gymnastics are applicable in a general sense, the following must be added as the most important for the treatment of heart diseases: The patient should be exhorted to overcome the resistance so slowly that his breathing will remain absolutely quiescent, or, as I usually express it, so that he shall have enough breath left in order to speak with ease at all times. The operator himself must observe the patient's mouth and nostrils carefully; any trace of commencing dyspnea is an indication for pausing until the breathing has become distinctly quiet again.

If necessary, a single movement may be divided up into several sections, between which the limb may rest while supported in the hand of the gymnast; following each single movement there should be a pause of one to three minutes' duration, and the patient may sit down to avoid the slightest tiring. At the end of the *séance* the patient should rest quietly on a couch for about fifteen minutes.

At the end of this article are numerous illustrations of the most important exercises, from which may be observed the positions and movements of the patient, and of the operator as well. Bedridden patients can, of course, carry out only a few of these exercises in the supine position. In other cases standing is difficult, so that those exercises only are possible which can be practised while in the sitting posture.

Whereas at the beginning the greatest possible care is necessary, as the treatment progresses more powerful resistance can always be withstood with ever-shortening pauses, as the heart itself becomes more vigorous from the exercises.

With young persons who are in process of rapid growth, and in whom the chest has not at the same time developed sufficiently, and likewise in persons with kyphosis and advanced scoliosis, the heart often remains weak, and, most frequently of all, the respiration is imperfect. It is of advantage to employ, in these cases, breathing gymnastics with deeper inspiration and expiration. So long as the bones and rib cartilages are still soft and elastic it is possible to produce expansion of the chest by means of such gymnastics, and to obtain an improvement in the respiration and in the pulmonary circulation, and, as a result, an invigoration of the heart also.

In cases of disturbance of the peripheral circulation, or where edema has already occurred, *massage* may be employed with good results. This consists best of all in centripetal stroking of the extremities, in the form of "*effleurage*" and "*petrissage*," in order to facilitate the return of the blood to the heart, and also the absorption of the edematous fluid. In cases in which there is increased rapidity of the heart's action, "*tapotement*," which is usually carried out on the body, together with the employment of an ice-bag, produces a rapidly sedative effect; this action, however, does not usually last very long. The same remark applies to vibratory massage, which is often employed nowadays instead of manual massage. The action of this vibration massage is also insufficient and of short duration, even when it is carried out in conjunction with high-frequency currents. This has been frequently confirmed from other quarters, as, for example, in a work recently published by Plate and Bornstein.

We have also recommended the *application of heat* in cases of weakness of the cardiac muscle, in which the heart requires a more rapid stimulation. Best of all is the use of hot water, in a rubber bag, at a temperature between 140° F. (60° C.) and 160° F. (71.1° C.), which should be applied lightly to the region of the heart.

The mechanical treatment of heart disease in the form of gymnastics and massage has been specially cultivated in Sweden. The fact that these methods, discovered and developed in an empiric manner, have clung to hard-and-fast rules, had confined their use to that country alone, until the work of Wide and others caused them to be slowly taken up by other nations.

The Swedish movements first obtained general dissemination through their talented advocate, Zander, who, along with manual treatment, employed also very cleverly constructed apparatus, which forms a distinct acquisition to our curative stores. Much that has already been described as to the action of resistance gymnastics applies also to the machine gymnastics of Zander. These apparatuses are intended to make the patient independent both of the gymnastic operator and also of his own physical condition. As to the latter statement, opinions are divergent.



A constant supervision is very necessary with machine gymnastics, as much on account of the regulation of the resistance as also to note the condition of the patient during and after the movements. But even with an exact control by the physician, it is impossible to equalize the imperfections which exist in the nature of the machine. If the resistance is at first made too great, the patient must endeavor to overcome this resistance during the whole period of the movement. An exact individualization, or even an increase or diminution during a single movement, cannot be attained; the machine does not adapt itself to the case. A resistance which at one time was correct may become far too great on the repetition of the movement, owing to the rapidly changing condition of the diseased heart. Machine gymnastics may, therefore, be a source of danger to the patient, and in fact overstraining is not uncommonly observed after their use. At the same time, Zander's machines are unquestionably a great advance on the many one-sided apparatuses, such as the ergostat and several others.

Some years ago, by the employment of a wheel mounted eccentrically, as well as by weights moving on inclined planes, Herz improved the apparatus of Zander; his apparatus was also considerably used. The expensiveness and need of management for Zander's and for Herz's apparatuses, and the circumstance that for their use the patients are limited to place and time, stand in the way of their general employment.

Oertel, who at one time gave a great impetus to the treatment of heart diseases, combined with his mechanical treatment a dietetic therapy, which consisted principally in a limitation of the quantities of fluid. We shall discuss here merely his mechanical method of treatment. Oertel employed in his method the movements of walking and climbing, and hoped that this increased muscular activity would produce an invigoration of the heart, and a removal of the circulatory disturbances. For these climbing exercises, Oertel selected mountainous regions—"*Terrainkurorte*," as he called them—which were suitable for his purpose by reason of their ascending paths. So long as it is a matter of treating young and muscularly strong persons, of a rugged physique, good results can certainly be obtained by this method; but with definite

heart disease, however, whether of an organic or functional nature, climbing is an uncontrollable form of gymnastics which cannot be administered in definite dosage, and which cannot be used at the beginning, but only at the end, of the treatment, when the heart has become so far invigorated through other methods of treatment that it can undertake such extra exertion as hill-climbing requires, without any actual danger.

### GYMNASTIC POSES FOR RESISTANCE EXERCISES.

Many of the resistance exercises have been described and illustrated in various publications, and here is presented on the pages that follow an orderly *résumé* of the various different movements devised by the originator of the method. The very practical plan has been adopted of exhibiting photographic reproductions of the actual gymnastic movements, as posed by two trained operators who have had long experience in this class of work. While these illustrations do not cover all the movements that can be executed, a quite general selection has been made in order to elucidate the subject as fully as possible.

The principles already laid down have been adopted by ourselves and accepted by our colleagues, and are, therefore, the present governing standards. We will, however, briefly recapitulate a summary of the more important regulations which govern these passive resistance exercises:

1. Passive resistance movements include abduction, adduction, flexion, extension, and rotation in a vertical, horizontal, or lateral direction.
2. These movements should so alternate that new groups of muscles are continuously made to act in sequence, thus avoiding fatigue.
3. The resistance should be made by the operator as slowly and gently as possible, but with as much firmness and muscular power as the patient's physical condition will warrant.

4. The operator should never grasp the patient's limb tightly, but should oppose its movement by firm counter-pressure against the advancing side, thus retarding the movement, but always permitting the patient to retain the "*balance of power*."

5. The operator should change his resistance whenever the direction of the physical force is changed.

6. To gain a well-balanced and uniform effect, these exercises should always be bilateral.

7. The operator should closely watch the patient's breathing and circulation, and at the slightest sign of embarrassment should stop the exercises. The patient should never be allowed to hold his breath while exercising.

8. A pause of one or two minutes should be allowed after each exercise in order to avoid any fatigue. The patient may sit down during the pause, especially during the latter half of the *séance*.

9. The length of time devoted to each *séance* should be about a half-hour. At the end of that period it will frequently be found that the number of heart-beats has been reduced from 10 to 15 per minute, and that the area of cardiac dullness has been made to contract an inch, more or less.

10. After the *séance* is finished, the patient should rest quietly on a couch for at least fifteen minutes.

This resting period is an essential detail of the Schott treatment that should never be abridged nor omitted. As a rule, the patient experiences a sense of moderate fatigue after the completion of the exercises, and in order to counteract this the medical attendant must insist upon from fifteen to thirty minutes of absolute muscular relaxation, with the subject lying prone on the back in a quiet darkened room until sufficient time has elapsed for the creation of a new feeling of bodily vigor not apparent at the beginning of the exercises herewith illustrated.

When the patient has acquired sufficient experience with the resistance exercises, as given by an expert operator (especially after returning to his home), he can train himself to imitate these movements by a scheme of "self-resistance."

This method requires the simultaneous contraction of muscles that are antagonistic to each other. All the rules previously laid down for passive resistance movements must be carefully observed, or physical injury to the myocardium may be the result of such neglect.

The illustrations of the resistance exercises which follow are self-explanatory, but a descriptive legend has been appended to each figure, thus presenting a detailed account of the various movements (Figs. 21 to 61 inclusive).



\* Fig. 21.—EXERCISE No. 1. (First movement.) The *patient* standing erect extends both arms directly forward at the shoulder level, with the tips of his fingers touching. The *operator* places his fingers on the outer side of the patient's wrist and his thumb on the patient's palm. The *patient* now swings his arms outward in a quarter circle until fully extended at right angles. The *operator* advances a step toward the patient and makes resistance on the outer aspect of both wrists until the movement is completed.

---

\* This series of illustrations, from Schott's *Balneogymnastic Treatment in Chronic Diseases of the Heart*, are used through the courtesy of P. Blakiston's Son & Co., Philadelphia, publishers of the American edition of this work.



Fig. 22.—EXERCISE No. 1. (Second movement.) The *operator* shifts his fingers to the palmar surface of the patient's hands and again makes resistance as the *patient* returns his hands to the primary position in front. The operator retreats a step backward to allow room for the patient's hands to come together.  
—PAUSE.—

Fig. 23.—EXERCISE No. 2. (First movement.) The *patient* standing, with hands at his side and palms against his body, raises both his arms outward and upward to the level of his shoulders. The *operator* makes resistance by pressing on the backs of the patient's hands with the palms of his own hands.







Fig. 24.—EXERCISE No. 2. (First movement completed.) The arms of the *patient* are now fully extended and the first movement completed to the level of his shoulders, while the *operator* still maintains his resistance.

Fig. 25.—EXERCISE No. 2. (Second movement.) The *operator* changes his resistance by placing his palms beneath the extended palms of the *patient*, while the *patient* returns his arms to the primary position at his side.

—PAUSE.—





Fig. 26.—EXERCISE No. 3. (First Movement.) The *patient* standing with his arms at his side raises his hands forward and upward to the level of his shoulders. The *operator* makes counter-pressure on the upper edge of the *patient's* wrists with his thumbs but substitutes his fingers as the movement nears completion.



Fig. 27.—EXERCISE No. 3. (First movement continued.) The arms of the *patient* are extended in front while passing upward from the horizontal position to a vertical position above his head. The *operator* continues to make resistance with his palms on the *patient's* wrists.



Fig. 28.—EXERCISE No. 3. (First movement completed.) The *patient's* arms have reached a vertical position. The *operator* is still opposing this movement by pressing his palms on the backs of the patient's hands.

Fig. 29.—EXERCISE No. 3. (Second movement.) The *patient* now reverses the movement and presses downward returning his arms to the primary position at his side. The *operator* changes his resistance by pressing against the lower edge of the patient's palms or wrists with the tips of his fingers.

—PAUSE.—





Fig. 30.—EXERCISE No. 4. (First movement.) The *patient* standing with his arms held at his side presses backward and upward. The *operator* standing behind resists this movement by pressure on the backs of the patient's wrists. When the limit of this movement is reached the reverse is begun and resisted by the operator who makes pressure on the front of the patient's wrists until the primary position is reached.

—PAUSE.—

Fig. 31.—EXERCISE No. 5. The *patient* standing rotates his left arm. The *operator* offers resistance by grasping the patient's wrist. The patient duplicates this exercise by rotating his right arm in the same manner.

—PAUSE.—





Fig. 32.—EXERCISE No. 6. (First movement.) The *operator* standing just back of the patient places one hand on the patient's shoulder and the other on his wrist. The *patient* standing with his arm at his side and his elbow fixed flexes his arm until the palm of his hand touches his shoulder.

Fig. 33.—EXERCISE No. 6. (Second movement.) The *operator* now offers resistance to the back of the patient's wrist as the *patient* extends his arm and returns it to the primary position. The patient repeats the same exercise with his other arm.

—PAUSE.—







Fig. 34.—EXERCISE No. 7. (First movement.) The *patient* standing with his hand against his side presses his right arm forward and upward without bending the elbow, gradually describing a complete circle and returning to the primary position. The *operator* places one hand on the patient's shoulder and makes resistance with the other hand on his wrist. He changes his resistance as the direction of the force changes.

Fig. 35.—EXERCISE No. 7. (Second movement.) The *patient* is returning his arm to the primary position. The *operator* has shifted his resistance to meet the changed conditions. The patient repeats this exercise by describing the same arc with his other arm.

—PAUSE.—





Fig. 36.—EXERCISE No. 8. The *operator* grasps the patient's wrist loosely with one hand and makes counter-pressure on the back of the patient's hand with his other hand. The *patient* extends his hand by an upward movement, and then flexes by a downward movement. The patient repeats the same exercise with his other hand.

—PAUSE.—

Fig. 37.—EXERCISE No. 9. (First movement.) The *patient* standing with one arm extended at right angles and the palm of his hand facing upward flexes his arm until his hand touches his shoulder. The *operator* supports the upper arm of the patient by placing one hand beneath it and makes resistance by pressing on the front of the patient's wrist with his other hand.





Fig. 38.—EXERCISE No. 9. (Second movement.) The *operator* supports the patient's elbow with one hand and makes counter-pressure on the back of the patient's wrist with his other hand as the *patient* returns his arm to the primary position. The patient repeats the same exercise with his other arm.

—PAUSE.—

Fig. 39.—EXERCISE No. 10. (First movement.) The *operator* stands at the right side of the patient and with his right arm extended across the patient's chest grasps the left shoulder with his right hand, and at the same time presses on the small of the patient's back with his left hand. The *patient* then slowly bends the trunk forward until a right angle is nearly reached.





Fig. 40.—EXERCISE No. 10. (Second movement.) The *operator* changes his right hand to the front of the patient's chest and his left hand to the patient's back between the shoulders, where he makes counter-pressure as the *patient* straightens up into the primary position.

—PAUSE.—

Fig. 41.—EXERCISE No. 11. (First movement.) The *patient* properly supported by the *operator's* hand on the back of his neck and the other on his chest, bends his trunk backward as far as possible.





Fig. 42.—EXERCISE No. 11. (Second movement.) The *operator* presses with his right hand on the patient's chest and his left hand on the small of the patient's back as the *patient* returns to the primary position.

—PAUSE.—

Fig. 43.—EXERCISE No. 12. (First movement.) The *patient* bends the trunk of his body sideways. The *operator* stands at the front of the patient with his right hand on the patient's chest under the left axilla and his left hand on the patient's right hip. The *patient* then bends his body toward the left side.







Fig. 44.—EXERCISE No. 12. (Second and third movements.) The *operator* now reverses his hands and makes counter-pressure as the *patient* bends his body toward the right side. The *operator* again reverses his hands and makes counter-pressure as the *patient* returns to the primary vertical position.

—PAUSE.—

Fig. 45.—EXERCISE No. 13. (First movement.) The *operator* standing in front places both hands on both shoulders of the *patient*. The *patient* then rotates his trunk to the extreme right side, while the *operator* presses against the left shoulder and pulls on the right shoulder, meanwhile stepping halfway around the *patient*.





Fig. 46.—EXERCISE No. 13. (Second and third movements.) The same movement is repeated by the *patient* turning toward the left side, the *operator* pulling on the left shoulder and pressing on the right. The *patient* again reverses and returns to the primary position, while the *operator* makes a reverse counter-pressure and steps back to his first station.

—PAUSE.—

Fig. 47.—EXERCISE No. 14. (First movement.) The *patient* standing rests one hand on the back of the chair while the *operator* stooping places right hand on the front of the patient's ankle and resists the forward position of the patient's foot.





Fig. 48.—EXERCISE No. 14. (Second movement.) The *operator* reverses his hand to behind the *patient's* ankle while the *patient* draws his foot backward to the primary position. The same exercise is repeated with the *patient's* other foot.  
—PAUSE.—

Fig. 49.—EXERCISE No. 15. (First movement.) The *patient* standing rests one hand on the back of the chair and extends his foot laterally, outward and upward, while the *operator* stooping makes resistance on the outer side of the *patient's* ankle.





Fig. 50.—EXERCISE No. 15. (Second movement.) The *patient* returns his foot to the primary position on the floor while the *operator* presses on the inner side of the patient's ankle. The same exercise is duplicated with the patient's other foot.

—PAUSE.—

Fig. 51.—EXERCISE No. 16. (First movement.) The *patient* standing with both hands resting on the back of the chair presses his leg backward and upward while the *operator* stooping makes counter-pressure on the back of the patient's ankle.





Fig. 52.—EXERCISE No. 16. (Second movement.) The *operator* changes his resistance to the front of the patient's ankle, as the *patient* returns his foot to the primary position. The same exercise is duplicated with the patient's other foot.

—PAUSE.—

Fig. 53.—EXERCISE No. 17. (First movement.) The *operator* stooping makes pressure on the top of the patient's foot while the *patient* standing rests one hand on the back of the chair and draws his foot directly upward.







Fig. 54.—EXERCISE No. 17. (Second movement.) The *operator* reverses his resistance by placing his hand beneath the sole of the *patient's* foot as the *patient* returns his foot to the primary position. The same exercise is duplicated with the *patient's* other foot.

—PAUSE.—

Fig. 55.—EXERCISE No. 18. The *patient* standing rests one hand on the back of the chair and rotates his leg to the right and left while the *operator* stooping grasps the *patient's* leg near the ankle. The *patient* repeats the same exercise with his other foot.

—PAUSE.—





Fig. 56.—EXERCISE No. 19. (First movement.) The *patient* seated in a chair presses one foot forward while the *operator* stooping makes resistance with his hand on the front of the patient's ankle.

Fig. 57.—EXERCISE No. 19. (Second movement.) The *operator* makes counter-pressure at the back of the patient's ankle as the *patient* returns his foot to the primary position. The patient repeats the same exercise with his other foot.

—PAUSE.—





Fig. 58.—EXERCISE No. 20. (First movement.) The *patient* seated in a chair presses his knees outward as the *operator* stooping makes counter-pressure with his hands on the outer side of each knee.

Fig. 59.—EXERCISE No. 20. (Second movement.) The *operator* changes his resistance to the inner side of each knee as the *patient* returns his knees to the primary position.

—PAUSE,—





Fig. 60.—EXERCISE No. 21. (First movement.) The *patient* seated flexes his foot as the *operator* stooping makes counter-pressure on the top of the patient's foot.



Fig. 61.—EXERCISE No. 21. (Second movement.) The *operator* changes his resistance to the sole of the patient's foot as the *patient* returns his foot to the primary position. The patient repeats the same exercise with his other foot.

—PAUSE.—

## BIBLIOGRAPHY.

1. Amer. Jour. Med. Sciences, June, 1915.
2. Bliss: Röntgen Diagnosis in Pathologic Lesions of the Teeth, Penna. Jour. of Röntgenology, January, 1917, p. 9.
3. Lewis: Mechanism of the Heartbeat, p. 266.
4. Arch. d. mal. du cœur, Feb., 1916.
5. Am. Jour. Med. Sciences, July, 1915.
6. Kuno: Journal of Physiology, 1915, p. 2.
7. Starling: Lancet, i, 569.
8. Robey: Amer. Jour. Med. Science, cliii, No. 4, 546.
9. From Carter's Diet Lists, Saunders, Phila., 1914.
10. Zentralblatt für Physiologie, No. 20.
11. Präger medizinische Wochenschrift, No. 43, p. 601.
12. Physiological Chemistry, Lecture XX.
13. Warthin: Am. Jour. Med. Sciences, October, 1916, No. 535, p. 508;  
also Transactions of the Association of the American Physicians, 1914,  
p. 416.
14. Howard: Johns Hopkins Bulletin, iii, 266.
15. Irving: Jour. A. M. A., lxiv, No. 13, 935.
16. Cohn: Jour. Exper. Med., xxi, No. 6.
17. Jour. Pharm. and Exp. Ther., iii, 89.
18. Jour. A. M. A., lxiv, 898.
19. Hall: Medical Times, xlv, 39.
20. Amer. Jour. Med. Sciences, cxlix, 696.
21. Jour. Pharm. and Exp. Ther., vi, 331.
22. Schott: Balneogymnastic Treatment in Chronic Diseases of the  
Heart. English Trans. by S. Lewis Ziegler, M.D., Phila, 1914.
23. *Ibid.*
24. Schott: Berliner klinische Wochenschrift, 1885.





# Diseases of the Respiratory System

BY

FRANK A. CRAIG, M.D.,

Instructor in Medicine, University of Pennsylvania; Visiting Physician,  
Henry Phipps Institute, University of Pennsylvania; Visiting Physician,  
White Haven Sanatorium; Physician in Charge of the Tuberculosis Class, Presbyterian Hospital, Philadelphia.



# Diseases of the Respiratory System.

---

## FOREWORD.

THE importance of the diseases of the lungs may be readily determined by a study of the mortality reports published by any large community, where it will be seen that the deaths due to diseases of the respiratory system, including pulmonary tuberculosis, far exceed those due to diseases of any other system.

In spite of the enormous annual loss from these diseases, they have not received the attention that their importance would warrant—with the possible exception of pulmonary tuberculosis. Even here the greatest progress has been along lines of prevention and the development of a more efficient method of applying remedial measures, rather than any distinct improvement in the actual diagnosis or treatment of the disease in the individual case. The increased interest in these diseases manifested in more recent years is probably to be accounted for by the fact that the Röntgen rays offered a newer method of study, and the diseases of the lungs have been more and more the subject of surgical treatment, which necessarily requires a very great accuracy in diagnosis. The Wassermann reaction and the salvarsan preparations have undoubtedly also stimulated interest in the pulmonary manifestation of syphilis, and recent studies of the chronic non-tuberculous diseases of the lungs have opened a new field of investigation.

The following pages do not contain any very radical changes in the methods employed for various diseases of the respiratory system. The effort has been made to present a brief review of the pathologic anatomy, diagnosis, and treatment of the more common conditions, avoiding as much as possible the various opinions held by different writers, and confining the work as much as possible to the views most generally accepted among the observers whose experience and

judgment would appear to make them best qualified to decide such questions. The sections on diagnosis and treatment are not intended to cover the entire field, but to describe such methods as have proved of practical value in recognizing disease, and the lines of treatment which have given the best results in my personal experience. Where I have felt that my experience was not sufficiently extensive to warrant the drawing of conclusions, the views of more experienced men have been accepted.

The work is written in the hope that it will present to the practising physician a study of the various diseases of the respiratory system which will prove of help in the handling of these conditions, which are so prevalent, and frequently so resistant to treatment.

### ACUTE AND SUBACUTE BRONCHITIS.

The treatment of this extremely common affection may be considered under several different headings as prophylactic, local, or general, the plan pursued in the individual case being dependent upon whether the condition is primary or secondary, and the portion of the bronchial system affected. A knowledge of the various causative factors and a general conception of the pathologic processes involved are essential for an intelligent management of the case.

The disease may be briefly described as a catarrhal process of the mucosa of the larger bronchi, the smaller bronchi, and the bronchioles, either alone or in various combinations. In the early stages it is characterized by a hyperemia of the membrane, which becomes reddened, swollen, and congested. In this stage the surface is usually covered with a small amount of mucopurulent secretion of a very tenacious character. With the development of the second stage the hyperemia diminishes, with an increase in the amount of secretion, which at first may be thick and tenacious, but later becomes more copious and less viscid, as the exudation from the blood-vessels and the secretion from the mucous glands become freer. With the progress of the disease the expectoration tends to lose its mucoïd character and become more purulent. Microscopically, the condition is characterized by an engorgement



of the smaller blood-vessels and capillaries, a leucocytic infiltration, and relaxation and swelling of the inner fibrous coat. The basement membrane becomes edematous and wrinkled, and a separation of the ciliated columnar epithelium occurs. The lumen of the smaller bronchi, which have become diminished by the hyperemia, may be partly occluded by the desquamated cells, leucocytes, and the secretion of the mucous glands. As the process becomes more severe the leucocytic infiltration may invade the deeper portions of the bronchial walls, as well as the muscular and outer fibrous coats. The secretion from the mucous glands becomes progressively excessive as the process develops, a process of desquamation usually affecting the epithelial and secretory elements of the glands.

The onset of the disease is usually sudden, with a feeling of chilliness, malaise, general pains, headache, and a sensation of rawness or tickling in the trachea. As the acute catarrhal process is seldom confined to the bronchial mucous membrane, the signs of coryza, pharyngitis, or laryngitis are usually associated with the symptoms of bronchitis.

The *signs* and *symptoms* of bronchitis correspond to what one would expect as a result of the pathologic processes in the bronchi. During the early stages the cough is hard, dry, unproductive, with substernal soreness and pain, a sense of suffocation or embarrassed breathing, and on examination sonorous or sibilant râles are found, when the smaller bronchi or bronchioles are affected. When the trachea and main bronchi only are inflamed, if the process is not of a severe type, physical signs may be entirely lacking on examination of the chest. As the second stage develops, the difficulty in breathing and pain may disappear as the secretion is coughed up with greater ease, the cough is not so incessant, and on examination the character of the râles becomes more indicative of moisture.

The chief cause of primary bronchitis is bacterial invasion, but even here there is usually some secondary factor which is indirectly responsible for the infection, such as irritation of the mucous membrane by various dusts, chemical irritants, noxious gases, smoke, and atmospheric conditions. There may be some lowering of the general resistance of the individual, such as follows sudden chilling, prolonged exposure to

cold and moisture, alcoholic excesses, and general diseases, or it may be merely an extension of a similar process in the upper air passages, such as coryza, tonsillitis, pharyngitis, laryngitis, and similar conditions. As a secondary process bronchitis accompanies many of the general infections, such as the acute contagious diseases common to childhood, influenza, typhoid fever, malaria, tuberculosis, and syphilis, and is commonly met with in persons suffering from various chronic diseases, such as cardiac affections, nephritis, gout, rheumatism, and the acute and chronic non-tuberculous diseases of the lungs.

In the majority of cases the micro-organisms directly responsible for the bronchitis are the influenza bacillus, the pneumococcus, streptococci, staphylococci, micrococcus catarrhalis, either singly or in various combinations. Less frequently other pyogenic bacteria have been found in the sputum in bronchitis, but the possibility of contamination of the sputum with germs from the mouth, in spite of careful washing and other measures to prevent such a source of error, makes one hesitate to accept many of the published reports upon the bacteriology of acute bronchitis. The micro-organisms mentioned are frequently responsible for the acute bronchitis accompanying the infectious diseases, such as typhoid fever and tuberculosis, although the bacteria causing the systemic infection may be the direct cause of the bronchial inflammation.

### TREATMENT.

The first step in the management of a case of acute bronchitis consists in the determination of any possible secondary cause, and its correction by appropriate treatment. The elimination of various occupational dusts, improvement of the general resistance to infections by the correction of errors in living conditions and change of climate, the improvement of various functions of the body impaired on account of functional or organic disease, all are important measures, especially from the standpoint of prevention.

Upon the first indication of the disease it may be possible to abort the attack by appropriate measures, such as absolute rest in bed (an extremely important measure), hot lemonade

or similar hot drinks, either with or without whisky, hot mustard foot-bath, and thorough elimination through the skin, kidneys, and intestinal tract. In this stage there is probably no medicine quite so valuable as Dover's powder in doses of 3 grains (0.19 Gm.), repeated hourly for three doses.

The general pains and malaise may be so severe as to demand special treatment, and there is probably no better method of relieving these symptoms than by the use of phenacetin in 3-grain (0.19 Gm.) to 5-grain (0.32 Gm.) doses every two or three hours. Aspirin may be used instead of the phenacetin, or combined with it, in doses of 3 to 5 grains (0.19 to 0.32 Gm.). The profuse sweating which occasionally follows the use of aspirin is not a disadvantage, but certain individuals have an idiosyncrasy toward this drug which may be manifested by generalized edema, chiefly of the face, ears, and neck. The saline purgatives are especially indicated in adults, while castor oil is more satisfactory in children. To derive any benefit from increased intestinal elimination, it is necessary to secure active purgation, with copious, watery evacuations, and this should be borne in mind in prescribing the purgatives, so that the dose may be sufficiently large to insure obtaining the desired results. Laxatives or purgatives in insufficient doses only add to the discomfort of the patient without any benefit being derived from their administration. The local application of heat to the chest not only relieves the sense of tightness and soreness, but seems to aid in relieving the congestion of the bronchial mucous membranes, promoting secretion and quieting cough. This is especially true in young children, in whom local measures alone will often serve to relieve completely all symptoms of bronchial irritation or inflammation. The local applications most commonly employed are mustard plasters, poultices, kaolin, and turpentine stupes (the fumes from which may prove irritating), or rubbing the chest with camphorated oil or camphor liniment may give more comfort and relief. The mustard plasters prepared by the commercial houses have the advantage of being light in weight, cleanly, easily applied, and may be moved readily from one part of the chest to another. Unless the home-made mustard plaster is carefully prepared, it may only add another discomfort to the patient. Many patients

derive considerable relief from rubbing the chest with liniment containing oil of gaultheria.

When the large bronchi are chiefly affected, relief may be obtained from the inhalation of a spray of bland oil to which has been added small quantities of menthol, eucalyptol, camphor, or creosote, the finely divided spray being directed toward the posterior pharynx, while the patient takes deep inhalations which are retained as long as possible. The inhalation of steam is also of considerable help, especially when impregnated with compound tincture of benzoin, sodium chlorid, sodium carbonate, creosote, or chloroform. While the various appliances for inhaling steam are very convenient, the use of a preserving-jar one-third full of boiling water to which is added the medication desired, serves the purpose very well, especially if surrounded by a towel which makes it more convenient for handling and also helps to direct the steam by fitting closely around the patient's mouth. The use of steam is of special value in young children, especially when the smaller bronchi are affected, when it may be found more convenient to use the croup-kettle and tent on account of the difficulty in having them use the inhaler. The use of the hot pack is also of considerable help in these young patients, particularly when the attack is severe and accompanied by considerable difficulty in breathing.

It is also necessary to pay attention to the general surroundings of the patient; to see that rest in bed is persisted in; to provide a sufficient supply of warm, fresh air; and to secure free elimination by the bowels, kidneys, and skin. While a light, easily digested diet is desirable, care must be taken to see that the patient receives sufficient nourishment, as it is of the greatest importance to have the general nutrition maintained.

In the early stages the drug which is of special value is potassium citrate, given in doses of 5 to 15 grains (0.32 to 0.97 Gm.) every hour or two to be of any value, to which may be added spiritus ætheris nitrosi in doses of 15 to 30 minims (0.97 to 1.94 mil). The increased elimination by the kidneys, skin, and intestines will usually be sufficient to relieve the bronchial congestion. When the cough is very "tight," with scanty, tenacious expectoration, it may be necessary to give,



in addition to the above, syrup of ipecac 5 to 15 minims (0.32 to 0.97 mil), or syrup of squill 15 to 30 minims (0.97 to 1.94 mil). Apomorphin in doses of  $\frac{1}{32}$  to  $\frac{1}{24}$  of a grain (0.002 to 0.0027 Gm.) may prove useful in some cases for increasing the amount of secretion from the bronchi, and rendering it easier to expectorate.

All emetics should be given in small doses, gradually increased, as many individuals are extremely susceptible to them, even small doses causing very distressing nausea, and perhaps vomiting. While antimony and aconite are used quite frequently in this stage, they should be used with extreme caution, on account of the possibility of their causing excessive depression.

It is in the early stage that opium or its derivatives are occasionally of value in controlling the incessant, hard, unproductive cough, but owing to the general tendency to employ opiates, especially heroin and codein, too freely in bronchitis, even in cases in which they are contraindicated, a word of caution might not be out of place. In the very young or the aged any opiate should be employed with extreme care, and never in cases in which there is much difficulty in breathing, or where the bronchioles are inflamed to any great extent; opiates are always contraindicated in the cases in which there is considerable secretion to be expelled. The field of usefulness of opiates in bronchitis is confined to those cases in which the cough is excessive and out of all proportion to the amount of expectoration, and even here it may be questionable as to whether the comfort obtained from their use is not counterbalanced by their tendency to check secretion, the stimulation of which is one of the objects of the treatment in this stage. When the danger of exhaustion from the constant, hard cough becomes imminent, and it is necessary to obtain some relief for the patient, it will be found that Dover's powders in small doses frequently repeated will be much more advantageous than either heroin, codein, or morphin. While Dover's powder only contains 10 per cent. of ipecac, one must be careful in prescribing an additional quantity of ipecac to avoid giving this drug in excess of the expectorant dose.

For increasing the quantity of the secretion and rendering it less tenacious, there is probably no drug which is of so



much value as ammonium chlorid in 5-grain doses (0.32 Gm.) every two or three hours. While certain vehicles commonly employed are soothing to the pharynx, it is inadvisable to administer expectorants in syrups, especially when given at frequent intervals, or over a prolonged period; peppermint water, cinnamon water, or some similar solvent will be found much more satisfactory in the average case.

When there is a copious tenacious secretion in the smaller bronchi and bronchioles, it may become imperative at times, especially in children, to secure active evacuation of the collected secretions, on account of the obstruction to breathing. Ipecac or apomorphin in emetic doses will usually bring about the desired result, the mucus being mechanically dislodged by the violent efforts of vomiting. It may be necessary to resort to the more stimulating expectorants, if the secretions should continue for any prolonged period. (See p. 336.) Occasionally the cough and expectoration are of a spasmodic character, in which case belladonna, stramonium and lobelia may prove of value; or the expectoration may be very copious, when, again, belladonna may be employed. At times the attack of bronchitis is accompanied by signs of prostration and cardiac weakness, especially in elderly people or those exhausted by prolonged illness, in which event prompt stimulation may be necessary. Many cases of acute bronchitis do not completely recover from the acute attack, and are left with a slight occasional cough, with more or less expectoration. Most of these are more benefited by tonic treatment and by insistence upon a rational mode of life than by any treatment directed toward the bronchial mucosa.

### CHRONIC BRONCHITIS.

An acute attack of bronchitis may be prolonged for a period of time so as to warrant the term "chronic bronchitis" being employed, and under this heading are included the cases of frequently recurring attacks of acute bronchitis. Bronchitis of a chronic type extending over a long period is very rare as a primary disease, and its occurrence should at once suggest that there is some secondary condition responsible for the persistence of the cough, especially when present in young

people. Continued exposure to irritating dusts and fumes may occasionally be responsible, but tuberculosis, chronic disease of the heart, chronic non-tuberculous disease of the lungs, nephritis, gout, and other diseases should always be suspected in these cases, and when present receive appropriate treatment. Recurrent "winter coughs" are not uncommon, appearing each year with the first damp, cold days of autumn, and continuing through the winter; these occur frequently in elderly, debilitated people, but may also occasionally affect the young.

In chronic bronchitis the changes found in the acute form are usually present in a modified form. The mucous membrane of the bronchi appears reddened, thickened, and ulcerated, or may be entirely lacking, the denuded surface of the muscular and fibrous layers being exposed. Microscopically, the leucocytic infiltration may invade the entire bronchial wall, even extending into the peribronchial tissues. Connective tissue may replace the cellular exudate, and atrophy of the tissue of the bronchial walls and desquamation of the epithelium is a not uncommon result. The mucous follicles may undergo ulceration, and there may be slight dilatation of the bronchi which have lost their elasticity.

#### TREATMENT.

The most important step in the treatment is to make sure that it is not secondary to disease in some other part of the body, and the next to make sure that there is no obstruction present to prevent the patient breathing through the nose. Nothing could be more conducive to frequent infection of the bronchial mucosa than being constantly exposed to the cold, dry, unfiltered air which results from mouth-breathing. The air should pass through the nose in order that it become warmed, moistened, and cleaned, a function which the internal nasal chambers are so well fitted to perform. Possible sources of infection should also be sought for, such as chronic sinus disease, tonsillar abscesses, and mouth infections.

The measures which promise the greatest benefit are those directed toward the general health of the patient, and while a change of climate is frequently of value, this it is not always possible to secure.

Treatment should be directed toward the relief of any disease which might have any bearing upon the bronchitis, as one could not expect much from medication for the relief of the bronchial symptoms so long as the causative factor persists.

Where there is evidence of cardiac weakness or valvular incompetence, it is necessary to apply such measures as will help to overcome the difficulty, depending upon the nature of the cardiac disease. For these cases rest, digitalis, atropin, and massage may be employed, according to the etiology and amount of the decompensation.

Where nephritis exists, such dietary changes as may be required should be instituted, together with such restrictions in the amount of salt or fluids ingested as may seem necessary from a study of the functional capacity of the kidneys. The bronchitis accompanying pulmonary tuberculosis is considered in detail in the section dealing with that disease. (See p. 446.)

The measures followed by the greatest benefit are those directed toward the improvement of the general health of the patient, and no detail of the patient's mode of life is too insignificant to receive the careful attention of the physician in charge, if he expects to make any headway in the treatment of this disease. A study of the patient's customary dietary should be undertaken, and such changes made as may be indicated. The clothing of the patient should be warm enough for comfort, but not so heavy as to induce a constant state of perspiration, most people finding the open mesh underwear more satisfactory than the woolen for this reason. It is much wiser to depend upon the outer clothing for warmth, as they are more easily changed to meet the requirements. In the winter-time warm or fairly hot baths may be employed, preferably just before retiring, or if taken during the daytime they should always be followed by a cool or cold sponge bath and thorough rubbing down with a rough towel. Sufficient fresh air should be obtained both day and night, and on wet, raw, or windy days if possible the patient should avoid going out, but if not possible should be thoroughly protected against the cold and moisture. Windy days are especially objectionable on account of the dust in the air. The living- and sleeping- rooms should be well ventilated, which does not necessarily mean that they must be cold, as it is possible to obtain a

constant supply of warm fresh air. When steam or hot-water heat is employed, it is especially necessary to see that fresh air is admitted to the room, and the intense dryness of the air from these systems of heating may be largely overcome by keeping large open vessels of water on the radiators. The bedclothing should be sufficient to keep the patient comfortably warm, but excessive coverings should be avoided. The discomfort resulting from the excessive weight of many bedcovers may be avoided in most cases by placing extra covers between the mattresses or beneath the lower sheet. Sleeping between blankets or sheets made of outing flannel may prove advantageous in very cold weather to those who suffer from cold extremities, or when patients prefer sleeping in a cold room or outdoors.

When the patient's financial resources permit, great relief during the winter months may be obtained by a sojourn in some of the southern resorts. It is impossible to lay down any fixed rules as to which patients will be benefited by a stay at the seashore, inland, mountains, etc., as each case seems to be a law unto itself, the special climate which seems to be best suited to the patient being determined by experience or experiment. Where there are secondary factors involved, the question of climate may be largely influenced by the nature of the secondary process.

The general tonics such as iron, quinin, strychnin, arsenic, and similar drugs are of considerable value in many cases, giving relief from the annoying symptoms of chronic bronchitis in many instances in which measures more especially directed toward the bronchitis have proved useless. Cod-liver oil is of value in many cases, even in the absence of coincident pulmonary tuberculosis. Considerable relief may be obtained from the inhalations and local measures suggested for the treatment of acute bronchitis. The cough is likely to be extremely annoying in the morning, for the mucus which has collected during the night usually requires considerable effort to dislodge. On rising in the morning a glass of hot water to which has been added 10 grains (0.648 Gm.) of sodium bicarbonate, 5 grains (0.32 Gm.) of sodium chlorid will frequently relieve the morning cough, permitting the secretions to be expectorated more easily. The efficacy of



this mixture may be increased in some cases by the addition of 10 to 15 minims (0.64 to 0.97 mil) of spirit of chloroform.

For the relief of the cough and expectoration in chronic bronchitis, terpin hydrate is probably one of the best drugs which we possess, but it must be given in full doses to obtain the desired effect. Terpin hydrate may be given in capsules containing 3 to 5 grains (0.19 to 0.32 Gm.) each three or four times daily, preferably after meals. The elixir of terpin hydrate is a very convenient form in which the drug may be prescribed, as it contains 2 grains (0.13 Gm.) of terpin hydrate to the dram, and it may be given in doses of 1 or 2 teaspoonfuls (4 or 8 mils) or even 3 teaspoonfuls (12 mils), if necessary. As it contains a high percentage of alcohol, it must be freely diluted with water, the powdered drug being preferable in any case in which the use of alcohol is contra-indicated. Creosote (see section on Tuberculosis) is also of a great deal of value, especially where the expectoration is purulent or fetid. When the mucus is tenacious and scanty, potassium or ammonium iodid in doses of 5 to 10 grains (0.32 to 0.64 Gm.), will aid considerably in rendering the mucus more copious, and relieving the cough. Other drugs which have been recommended in chronic bronchitis are oil of cloves, oil of sandalwood, oil of eucalyptus, oil of turpentine, oil of copaiba, balsam of Peru, balsam of tolu, terebene, tar, and a number of similar drugs. The great disadvantage which practically all of these stimulating expectorants possess is their tendency to cause derangement of the stomach if continued for any length of time; for this reason ammonium chlorid, terpin hydrate, and creosote are the drugs upon which the main reliance will have to be placed in the treatment of this chronic process.

As a prophylactic and remedial agent in both acute and chronic bronchitis, bacterial vaccines have been administered in many cases during recent years with varying success. The main objection to this method of treatment lies in the difficulties attending the determination of the micro-organisms responsible for the infective process in any given case. In spite of the various measures suggested for collecting and washing the sputum, it is impossible to tell which bacteria are actually causing the inflammation of the bronchial mucosa,



even were it possible to eliminate absolutely the possibility of contamination of the expectoration by mouth bacteria. The preparation of an autogenous vaccine is a very unsatisfactory procedure, as it is impossible to tell whether the important micro-organism in a given case has been included in the vaccine or not, and stock commercial vaccines are open to the same objections in a more marked degree. It is, therefore, not to be wondered at that the results obtained by vaccine therapy should vary so greatly, and it makes it much more difficult to determine the actual value of such a method of treatment.

In a patient who is subject to repeated attacks of acute or chronic bronchitis, where other methods of treatment have proven unavailing, it may be worth while trying to secure a bacterial vaccine prepared from bacteria recovered from his own sputum, especially when the washed sputum has repeatedly shown, in predominant numbers, the presence of certain micro-organisms of recognized pathogenicity. While our present knowledge indicates that the injection of these bacterial vaccines are unaccompanied by any harmful effects, it would seem wiser not to use them indiscriminately in the absence of special indications, until further investigation has shown whether they are absolutely without danger or not.

### FIBRINOUS BRONCHITIS.

This relatively rare form of bronchitis may occur as a primary disease, but it is more frequently met with as a secondary process in some disease of the lungs or bronchi. Cases have been observed in patients suffering from pulmonary tuberculosis, asthma, pneumonia, diphtheria, typhoid fever, measles, scarlet fever, actinomycosis, chronic passive congestion, and as a result of the inhalation of steam, ammonia fumes, and smoke. The fact that the condition occurs in the course of so many different diseases would make it appear as if the predisposing cause was not peculiar to any special morbid process, and that the fibrinous bronchitis was merely incidental in its occurrence and not an integral part of the associated disease.

Fibrinous bronchitis is characterized by the formation within the lumen of the bronchial tubes of casts composed of

fibrin and mucin in varying proportions. The casts develop in different parts of the bronchial tree, and are expectorated from time to time after violent paroxysmal attacks of coughing. The disease may occur in an acute form with the formation of large casts occupying a considerable portion of the bronchial system, in which case severe dyspnea, relieved only by the coughing up of the large fibrinous casts, may accompany the attack.

The pathology of the process is obscure, and while at one time the view was held that denudation of the bronchial mucosa was essential for the fibrinous exudate to collect in the bronchi, it has been shown that casts may form in bronchi whose mucosa is intact. The nature of the process and the local conditions necessary to permit or to cause this exudation of fibrin through the mucosa of certain portions of the bronchial system is absolutely unknown. While bacteria have been supposed to play a prominent rôle in the process, this relationship has never been proved, and all efforts to produce the condition experimentally by the intra-bronchial injection of bacteria have so far been unsuccessful.

The *symptoms* of the acute and chronic cases, consisting of cough, dyspnea, and the expectoration of casts, are practically the same, although they differ markedly in severity. In the acute form the cast is usually larger than in the chronic type, the dyspnea and cough are prone to be much more severe, and may last for one or two days before the cast is expectorated. While cases have been reported which terminated fatally from asphyxia due to occlusion of the bronchi by large casts, this accident is extremely rare. The chronic forms are not so severe as the acute, and not attended with such severe cough or dyspnea. The casts may be so small that there may be no dyspnea, and the expectoration of the small plugs may not be accompanied by an unusually severe cough. The casts may be expectorated at intervals of varying length over a considerable period of time.

The *physical signs* vary with the size of the cast and its location. When large casts lodge, there may be restricted movement of one side of the chest, and the signs of bronchial obstruction are present over the area of the lung supplied by the obstructed bronchi. Dullness may be present, but usually

percussion is negative, and there are diminished or absent breath-sounds over the area affected, the breath-sounds returning after the cast has been expelled. Sonorous râles, usually localized, may be present, and a peculiar flapping sound has been described as occurring in certain cases due to the partial detachment of the bronchial cast.

The casts vary from small white masses, only recognizable as of bronchial origin when floated in water, to large, branched masses with the typical bronchial arrangement, several inches in length. They are white or grayish-white in color, and on section it may be seen that the thicker branches show a concentric laminated arrangement.

#### TREATMENT.

The treatment of the attack consists of attempting to aid in the freeing of the cast and its expulsion by means of sprays of lime-water and steam inhalations. Apomorphin hydrochlorate hypodermically has been suggested to assist in expelling the cast. The only drug which seems to be of any value internally in the treatment of the chronic cases is the iodide of potassium in large doses, and instances have been reported in which the administration of the drug has been followed by complete recovery. In those cases in which the dyspnea is severe the signs of impending suffocation may necessitate an attempt to remove the cast by bronchoscopy as a means of preventing a fatal termination.

When the process accompanies some other disease, this naturally should receive active treatment, and the usual measures for ordinary bronchitis should be employed, for the process is almost invariably accompanied by evidence of a general bronchial catarrh.

#### BRONCHIECTASIS.

Dilatation of the bronchi is a condition which varies very much in its general characteristics and etiology in different cases. While it may occur rarely as a primary disease, usually it is secondary to some other process, the generalized bronchiolectasis of childhood coming nearer to what may be classed as a primary disease than any other type, although

even here usually there is a history of preceding bronchitis. The causes may be briefly stated as: (1) changes in the bronchi themselves, such as loss of tone in the walls, stenosis, or obstruction of the bronchus; (2) changes in the lung, such as collapse, pneumonia, fibrosis, or emphysema; (3) changes in the pleura, such as compression of the lung due to effusion of long standing, or the extension into the lung of a fibrosis originating in the pleura. Thus bronchiectasis may result from acute bronchitis, tuberculosis, syphilis, foreign bodies, new growths, aneurisms, pulmonary cirrhosis or fibrosis, lobar or broncho-pneumonia, chronic pneumonia, pleurisy, or empyema. When it accompanies tuberculosis it may affect the bronchi of the upper portion of the lung, although the lower lobes are the parts usually implicated. Only rarely is the course of one bronchus implicated, the process usually being multiple or diffuse.

The bronchiectasis may occur as a uniform cylindrical dilatation of the bronchi, affecting the larger branches only, or extending to the smaller ramifications. Certain portions of the lung or lungs may share in the process, the remaining bronchi showing no abnormality, or one entire lung may be affected, but only very rarely does the process implicate both lungs in a generalized uniform dilatation of the entire bronchial system. The dilatation is uniformly cylindrical in only an extremely small proportion of the cases, nearly every case presenting constrictions of the bronchi here and there throughout their course. The saccular or globular type may occur in association with the above, or as a single process. In this form certain areas of the lung, which may be of considerable extent, are occupied by numerous small, rounded saccules varying in size, and usually found to communicate with the bronchi. The lung tissue in these cases usually is very much diminished, or may have completely disappeared, being replaced by fibrous tissue of varying density.

Microscopically, the changes are similar to those found in acute or chronic bronchitis, depending upon the stage of the disease, the process as a rule being more intense and extensive than in bronchitis. Thus, in bronchiectasis it is customary to find the connective tissue proliferation invading the entire bronchial wall and surrounding tissue, with compression and

atrophy of the normal muscular and fibrous tissues of the bronchus. In some instances all semblance to bronchial wall is destroyed, the tube being surrounded by a thick mural partition of dense connective tissue.

The exact nature of the pathologic process underlying the production of bronchiectasis is still an unsettled question, but whether due to factors within the bronchi, such as increased pressure, or to conditions in the surrounding tissues, such as traction and localized compression from contracting connective tissue arising in the lung or pleura, it must be conceded that an affection of the bronchial walls themselves is the essential factor. This change in the bronchial walls in many cases appears to be the principal cause of the lesion, and in some cases it may be the only one detectable. The exact nature and mode of operation of this process is not very well understood.

From the standpoint of diagnosis the *symptoms* usually are more characteristic than the physical signs, and the diagnosis may at times be made upon the symptoms alone. Among the most important subjective signs is the type of cough and expectoration so common in these cases, namely, fairly long periods during which there may be slight cough, or none at all, terminating in acute attacks of cough, with the expectoration of mucopurulent, frequently offensive material which varies in amount, but is commonly excessive. This accumulation and putrefaction of the bronchial secretion over long periods of time indicates in the majority of cases a dilatation of the bronchi, the material only being expectorated when the cavity has become overfilled or when some change of position has caused it to flow into the adjacent bronchi. The periods between the attacks may vary from a few hours to several days, or even longer, depending upon circumstances. At times this evacuation of the bronchial cavity is accompanied by a sudden violent expulsive effort, large quantities of foul-smelling pus being forcibly expelled from the patient's mouth or even from the nose. The sputum is usually mucopurulent, but may consist of pus only, and at times may show a tendency to separate into three layers, as described under Pulmonary Abscess (*q.v.*). It may be odorless, but is usually stale or musty, and may be exceedingly foul, the odor being



also given off by the breath. In one patient under the care of the writer the odor of the breath was so exceedingly foul and nauseating as to make it necessary to isolate the individual from the other patients in the sanatorium.

Hemorrhages, usually small in amount, may occur, and are due to vascular outgrowths or small ulcers on the bronchial walls, but large and even fatal hemorrhages have been reported as a result of rupture of branches of the pulmonary artery by erosion of the bronchial wall. The other symptoms which may be present are those referable to the general



Fig. 1.—Pulmonary osteo-arthritis. Thickening of the distal phalanges and curving of the nails in long-standing bronchiectasis.

toxemia resulting from the accumulation of pus, or to the bronchial, pulmonary, or pleural conditions responsible for the bronchiectasis.

The *physical signs* are to a great extent influenced by the associated disease process, as bronchiectasis is infrequent in a primary form. During the early stages the general health of the subject may not be affected, and there may be no elevation of temperature as long as the cavity is frequently evacuated. Hypertrophic pulmonary osteo-arthritis is more frequently present in bronchiectasis than in any other pulmonary disease, and the clubbing of the fingers and toes may be extreme. The drumstick appearance of the fingers, due to the enlargement of the distal phalanx, may be the only

evidence of the disease, or all of the phalanges may be clubbed, and even the distal extremities of the bones of the wrist and ankles, the changes being due to hypertrophy of the periosteum and the formation of new bone.

The examination of the chest in uncomplicated cases may reveal signs of a cavity when the contents of the dilated bronchi have been expelled, or there may be only scattered areas of impairment on percussion and localized râles. These may be bubbling, with a metallic, resonant quality, or merely fine, moist, and crepitant, due to the secondary changes in the surrounding pulmonary tissue. The signs of cavity may be elicited by placing the patient in the horizontal decubitus, or in the Trendelenberg position. When the process is deep-seated there may be no physical signs evident on the examination of the chest, even in cases in which all the symptoms and general appearance of the patient indicate that dilatation of the bronchi is present. As previously stated, the associated changes in the pleura or lungs may be of such a character as to obscure the signs of dilated bronchi on examination of the chest.

The *x*-ray examination may be employed with advantage in some cases, especially when the physical signs are obscure. The changes in the shadows observed in plates taken before and after the evacuation of the accumulated fluid is extremely suggestive in some cases.

Anyone who has seen these distressing cases in the advanced stages, in which the odor of the breath is so foul and nauseating that they cannot associate with anyone, expectorating large quantities of foul-smelling pus at frequent intervals, cannot help but be impressed with the futility of our present methods of treating this condition.

#### TREATMENT.

The most important step is the improvement in the patient's general condition by means of tonics, nourishing food, rest, and fresh air, as outlined under Tuberculosis. An effort should be made to correct any chronic bronchitis that may be present by means of creosote, terpine hydrate, oil of sandalwood, oil of cloves, and similar stimulating expectorants. The direct application of healing, antiseptic substances

has been attempted by means of inhalations and intratracheal injections, with the object of rendering the expectoration less purulent, and overcoming putrefactive changes in the accumulated material in the bronchi. The fumes of turpentine may be employed by placing a teaspoonful (4 mls) of the oil in a pint (500 mls) of boiling water, an improvised inhaler for which may be made by means of a quart jar surrounded by a towel. Creosote, oil of eucalyptus, carbolic acid, and thymol may be used in a similar manner. Various drugs may be employed in the form of a spray, or the fumes inhaled when they are volatile, and for the latter a mask may be employed upon which are placed such drugs as are indicated. The mask for providing medicated inhalations has never proved very popular in this country, the discomfort of wearing such an appliance being hardly compensated by the results obtained. The mask is only suitable for use during the daytime when the patient is confined to the house, or in a sanatorium or hospital, although there is no objection to its being worn at night, when a certain amount of benefit may be obtained in the few hours during which it is worn. A popular formula for a mixture to be employed in this manner is equal parts of carbolic acid or thymol, rectified spirits, and glycerin, 10 drops (0.6 mil) of the preparation being dropped upon the face mask and renewed as required.

A method of treatment very strongly recommended consists in placing the patient in a closed room in which creosote is heated in a metal saucer by means of a spirit-lamp. It is necessary first to protect the patient's hair and clothing by suitable impervious coverings, to plug the ears and nose with cotton, and to wear goggles over the eyes. The dense fumes of the creosote quickly fill the room, the patient being exposed to them for about fifteen minutes on alternate days at first, then every day, the time of exposure gradually being prolonged as the patient becomes accustomed to the fumes— even up to an hour and a half twice daily. The effect at first is to start up a severe cough, with a marked increase in the amount of expectoration, but the severity of the cough diminishes as the patient grows hardened to the treatment. While strongly recommended in this condition, it must indeed be a persevering patient who would continue such an unpleasant

method of treatment, unless marked improvement followed the ordeal very soon after it was instituted. The modification of the above may be used by floating the creosote on water in a small pan under which the burner is placed, thus affording steam creosote inhalations instead of the pure fumes obtained by the other method. The steam inhalations are borne very much better by the majority of patients.

The direct application of remedial agents to the bronchi has been carried out by means of intratracheal injections. These are made by means of a syringe with a long, curved nozzle, and cause the patient very little discomfort, if care is exercised in making the injection to have the tip of the nozzle well beyond the larynx, and the amount injected does not exceed 1 dram (4 mils). The fluid injected may be directed toward either lung by having the patient lie upon the corresponding side after the injection is made. The drugs are usually combined with olive oil as a vehicle, and the mixture warmed before use. Menthol, iodoform, eucalyptol, creosote, guaiacol, and silver and iodine compounds have been employed in this manner. A formula which has been recommended consists of menthol 10 parts, guaiacol 2 parts, and olive oil 88 parts. Theoretically one should expect to derive a great deal of benefit from this form of treatment; that the results obtained are not as satisfactory as one would anticipate may be inferred from the fact that this method of treatment has not been more generally adopted.

Bacterial vaccines have been recommended in the treatment of this condition, and with encouraging results in some cases. The objection to this plan of treatment is the same as in the case of bronchitis, but to a greater extent. The pus expectorated from these cases is very foul, and contains numerous varieties of micro-organisms, many of which can have no relation to the disease from the standpoint of etiology, being merely secondary contaminations.

Where the condition is due to actual primary changes in the bronchial walls, to fibrosis of the lungs, or to the pressure of new growths and aneurisms, very little can be done in the way of radical treatment. The best that can be expected in such cases is the amelioration of symptoms, even those due to syphilis, tuberculosis, and foreign bodies in the bronchi, in



which there is a possibility of removing or curing the causative factor, if the gross changes in the bronchi are very marked or extensive, the possibility of curing the condition by medicinal measures is exceedingly remote.



Fig. 2a.—Posture: The use of the foot of the bed.

Considerable comfort may be given these patients by relieving the cough and expectoration, and this can be accomplished by teaching them to assume for a short period several times a day the postures which facilitate expectoration. Thus a patient with dilated bronchi in the lower lobes may secure a very comfortable day if made to lie for from fifteen to thirty



minutes every morning with the feet much higher than the head, a position easily obtained by the elevation of the foot of the bed. He should lie flat on the back, face, or on either side, depending upon which position seems to facilitate the



Fig. 2b.—Posture: Inverted position, use of chair. (Kindness of Dr. A. H. Garvin, Dr. H. W. Lyall and M. Morita.)

evacuation of the accumulated expectoration. This same procedure carried out at night will usually secure a comfortable night's rest. The object is to promote drainage from the bronchiectatic cavities, thereby preventing the accumulation of pus, and the unfavorable chain of symptoms resulting from

the absorption of its products. A little experimentation will soon decide the posture which seems to promote this evacuation most readily; it may even be necessary for the patient completely to invert the body—a position which can be assumed by flexing the body at the waist over the foot of the bed or the back of a chair, with the extended hands resting on the floor. (Figs. 2a and 2b.) Where feasible, an operating table may be employed to place the patient in the Trendelenberg position, the subject lying either on the face or back as indicated. While most cases derive considerable benefit from this postural treatment when performed two or three times a day, in some it will be necessary to have them carry out the procedure every hour or two. The inverted position should be maintained for at least fifteen minutes on each occasion, or until complete evacuation has been secured, or the discomfort attending such a position proves too severe. This method is especially suited to those cases in which the bronchiectasis affects the bronchi below the level of the root of the lung, inasmuch as when the cavities are above this point the possibility of constant drainage is naturally much greater. The postural treatment of bronchiectasis has been followed in some cases by striking results, and should be tried carefully and conscientiously in every case. That it can be carried out by the patient himself, is without danger, and is easily secured by anyone, are recommendations in its favor.

Artificial pneumothorax has been recommended in the treatment of certain cases where the pleura is not markedly adherent, and in which the gross changes in the lungs are not extensive or very dense. Within recent years some of these cases in which the lesion is limited and localized to one lower lobe, surgical interference has been suggested, with the amputation of the diseased portion of the lung, or subperiosteal resection of the ribs. The field of pulmonary diseases has so recently been invaded by the surgeons that it would probably be the better plan not to recommend this method of treatment at the present time. In the future more care in the selection of cases and further operative experience may provide a surgical treatment which may prove of value in treating what at present must be looked upon as an almost hopeless condition.

Finally, when persistent treatment has failed, as it so frequently will in this disease, it may be advisable to advise a change of climate, that last resort of the discouraged and perplexed physician.

### BRONCHIAL ASTHMA.

The older writers on this subject described under the name asthma many varied conditions, leading to a certain amount of confusion, which is still apparent at times in modern literature. Formerly it was the custom to apply the name asthma to any condition which was accompanied by spasmodic attacks of dyspnea, which was usually qualified by the addition of the name of the associated condition, giving rise to such terms as cardiac asthma, renal asthma, etc. For many years there was considerable discussion as to whether there was such a disease as asthma which could occur as a primary disease, or whether the condition was merely a symptom of some other process. While the exact nature of the process is still somewhat obscure, it is generally recognized that bronchial asthma may exist as an essential disease process, and that while spasmodic attacks of dyspnea may occur in the course of certain diseases, which closely simulate bronchial asthma, they are not identical conditions.

The symptoms are due to a swelling and hypersecretion of the mucous membrane of the bronchi, with possibly a certain amount of spasmodic, muscular contraction. Whether this may come about through nervous derangement alone, or whether it is always a manifestation of anaphylaxis, is still open to question. The more recent view that asthmatic attacks are an indication of intoxication by certain substances to which the asthmatic individual has become sensitized is the most plausible which has been suggested, even if the exact *modus operandi* is still uncertain, and some of the cases strongly suggest a nervous origin. There are no clear-cut or characteristic gross pathologic findings in asthma, the few cases which have come to autopsy during an attack showing merely a redness of the bronchial mucosa, with slight dilatation of the bronchi, and more or less emphysema. The medium and smaller bronchi are usually filled with mucus, which contains epithelial cells, granular material, leucocytes, and the various special features described under the sputum. The microscopic changes in the bronchi are in no way characteristic, with the pos-

sible exception of the findings of eosinophiles and Charcot-Leyden crystals in the bronchial walls, and occasionally an increase in the amount of elastic tissue.

When present, the *symptoms* preceding the attack vary greatly in character, some patients feeling unusually well, others being depressed, and many presenting the signs indicative of a "cold," with general catarrhal symptoms. Occasionally the individual may be warned of the oncoming attack by the appearance of peculiar prodromal symptoms, such as voiding large quantities of urine, epigastric distress, or sweating. The attacks usually come on very suddenly, frequently within a few hours after going to sleep, and begin with a hard, dry cough, and difficulty in breathing, which becomes more and more severe, until frequently the patient feels as if death were impending from suffocation. The patient sits up in bed, with the windows wide open, literally gasping for breath, and in spite of the most violent muscular effort finds it almost impossible to get air into or out of the chest. What little interchange of air occurs is accompanied by loud, wheezing sounds, which usually can be heard at a considerable distance from the patient. Cyanosis is usually present, and frequently the skin surface is cold and covered with perspiration. Not only inspiration is extremely difficult, but expiration also, and frequently the expiratory difficulty is the most marked. The distended chest and diminished respiratory movement of the thorax, in spite of the violent efforts, present a very characteristic appearance on inspection.

In addition to the labored respiration and visible evidence of deficient aëration, examination of the chest shows soft or inaudible breath-sounds, their place being taken by wheezing râles; expiration is markedly prolonged, and the râles are seemingly more marked during this phase of respiration. The inspiratory murmur may be present, softer than normal, and only the expiratory sound obscured by the wheezing râles. Percussion is usually negative, although there may be a certain amount of hyper-resonance present.

Expectoration usually does not appear until near the end of the attack, the sputum as a rule consisting of small, rounded, hyaline granules or balls, translucent and grayish in color, these characteristic *perles* usually being mixed with more or less mucus. Microscopically, these small sago-like granules are seen to have



a thread-like structure arranged in a corkscrew manner—the so-called Curschmann's spirals. The spiral arrangement may be visible even to the naked eye, and is easily demonstrated under the low power of the microscope. Other distinctive features are the presence of Charcot-Leyden crystals, and a marked increase in the proportion of eosinophiles.

### TREATMENT.

The treatment of the cases of dyspnea which simulate bronchial asthma must be directed toward the associated heart, kidney, or pulmonary disease responsible for the development of this symptom. True bronchial asthma, as previously noted, may develop as a result of sensitization to some foreign protein, or may appear as a reflex nervous manifestation from the presence of some morbid condition in another part of the body. Polyps, or malformations and hypertrophic conditions in the nose, may be the exciting factors, also post-nasal adenoid overgrowth, and abnormalities of the larynx, trachea, and genitalia. The asthmatic attacks have been checked by correction of these abnormalities, in many cases being followed by an apparent cure. The first step in treating bronchial asthma between the attacks consists of the correction of any abnormality of the upper air passages or genitalia which may exist, in the hope that the case under observation may be of the reflex nervous type. The cases which represent anaphylactic phenomena are due to sensitization to a protein substance, which may arise from some latent focus of bacterial infection, such as chronic nasal sinus infection, alveolar abscess, and infection of the gall-bladder or of the bronchi themselves. The focus of infection should be sought for, and removed as soon as possible, in the hope of preventing the occurrence of further attacks. When the attacks result from intoxication due to sensitization with protein substances arising without the body, the problem is more complicated.

Asthma may result from sensitization to the pollen of certain weeds or grasses (the so-called hay-asthma), the emanations from horses or other animals, or from the ingestion of certain foodstuffs, such as eggs, shell-fish, or oatmeal, to mention only a few of the numerous sources of the protein which possibly may be responsible for the attacks. To determine the source of the offending protein in the individual



case may be attended with considerable difficulty, although a carefully recorded history or close observation by the patient may suggest the probable nature of the conditions which are necessary for bringing on the attack. The study of asthma from the standpoint of its relation to anaphylaxis has not continued for a period of time of sufficient length for the accumulation of the necessary data to render the treatment of the disease from this standpoint of that practical importance which it will undoubtedly reach in the future.

The cutaneous or intracutaneous (not *subcutaneous*) tests for sensitization to certain food proteins or animal proteins, with the subsequent employment of the specific proteins in the treatment of the disease, is still in its infancy. These tests for sensitization may be applied in the following manner: The history of the patient usually indicates to which group the protein to which they are sensitized belongs, whether the pollens, animal emanations, foodstuffs, or bacteria. By applying to the skin the various proteins belonging to that group it is frequently possible to identify definitely the exact protein responsible for the attacks. For example, if certain foodstuffs are suspected from the history, by careful questioning and observation one may be able to narrow down the possible proteins to those derived from meats. The soluble proteins of the various meats are then secured, either by making an aqueous extract of the meats, or by dissolving in sterile water the dried proteins which are now obtainable in a convenient form from the druggist. The skin of the forearm is then denuded of its superficial layers by means of a von Pirquet borer at a number of points, corresponding to the number of tests it is desired to make. To one point is applied normal salt solution or a 3 to 5 per cent. lactose solution for a control, and to each of the other points one of the protein solutions, care being taken to identify the protein applied to each point. In the case in question the solution of each meat protein should be given a number which should be marked on the skin, at some distance from the abrasion by means of a blue pencil. Care must be used in abrading the skin not to draw any blood or serum, merely the superficial layer of the skin being removed. The protein to which the patient is sensitized may be recognized by the development of a positive reaction at the point where it has been applied. This reaction is characterized by the development of a well-defined urticaria-like

wheel surrounded by a zone of erythema; it appears within from five to ten minutes, and may last for from one to three-quarters of an hour. A slight swelling not infrequently develops from the abrasion of the skin, but this false reaction should not lead to any confusion, as its nature may be recognized by the control test, which also will show the same phenomena.

The test may also be made by injecting a minute quantity (0.01 to 0.02 mils of a 1 or 2 per cent. solution) of the protein directly *into* the skin (not *beneath* the skin) by means of a very fine hypodermic needle (27-gage). The reaction is the same as by the other methods, and while it is more delicate than the cutaneous test, it usually takes longer for it to appear, is slightly painful, and there is a chance of the solution being injected beneath the skin, with the development of a general reaction, which in a very sensitive patient may lead to very alarming symptoms, and possibly to death. For this reason the intracutaneous test should be performed only by one accustomed to this method of application, the cutaneous test being much more preferable for general use. It is to be hoped that further study will simplify the methods now employed for determining the protein to which the individual has become sensitized, and the securing of desensitization, and will shed some light upon the problem of why only certain individuals should become sensitized to such proteins. The entire question of anaphylactic phenomena is one about which we know relatively very little at the present time.

The thorough study of a case of bronchial asthma between the attacks is a far from simple matter, but is absolutely essential in the majority of cases, if one expects to secure for the patient any permanent relief. A careful study of the nose, throat, ear, teeth, and in fact the entire body for possible abnormalities, defects, or sources of infection may be necessary. The sputum also should be carefully studied, with the hope of determining whether the constant presence of any certain bacteria in predominant numbers in the washed sputum suggests their bearing an etiologic relation to the process. Certain strains of streptococci, pneumococci, and fusiform anaerobic bacteria have been described as the probable cause of certain cases of asthma, and the experiments with the micro-organisms in some of the cases seem to support such views.

When the relation between the vegetable, animal, or bacterial protein and the asthmatic seizures can be definitely and positively established, the treatment of the patient may be cautiously begun by giving small quantities of the offending protein.

Desensitization may be accomplished in the case of food proteins by feeding the patient a small quantity of the offending food daily, gradually increasing the amount until the skin reaction fails to develop or becomes very weak. It is necessary to continue the ingestion of a moderate amount of the food, otherwise the patient may become sensitive again. Where the patient is extremely sensitive it may be necessary to begin the treatment with an exceedingly minute dose, which, if well borne, may be rapidly increased, the amount again being reduced upon the appearance of any toxic symptoms.

For desensitization to the other types of protein the subcutaneous injection of the protein may be employed, but for foodstuffs the administration by mouth is to be preferred. Great care must be used in the hypodermic method, as serious anaphylactic phenomena may follow the injection of too large a dose in a very sensitive patient. An initial dose of 0.00001 milligram of the various foodstuffs is safe to use in nearly every case, this initial amount being gradually increased. Care must be taken in increasing the dose to avoid causing any toxic symptoms, and if any should appear the subsequent dose should be well below that producing the unpleasant symptoms, and the following dose being more cautiously increased. The treatment may be continued until there is no longer a positive cutaneous test obtainable. The hypodermic injections should be given under complete aseptic conditions, using every precaution to avoid infection.

Caution must be employed in administering diphtheria antitoxin to asthmatics, as death has followed the injection in numerous instances. Whenever it may become absolutely necessary to employ diphtheria antitoxin in asthmatics, a preliminary skin test should be made to determine the patient's susceptibility before injecting any large quantity of the serum. This is especially true when the asthmatic attacks have been shown to bear a definite relation to horses.

The employment of bacterial vaccines in asthmatics must be used with some caution, for the condition may be aggravated by their use, although many writers have had very gratifying results from this form of therapy, especially when autogenous vaccines have been employed. The use of these products in this disease is open to the same objection which holds true in all broncho-pulmonary diseases, namely, the difficulty in determining definitely the organisms causing the disease process.

Certain observers have reported very gratifying results from the subcutaneous injection of autogenous defibrinated blood, obtained preferably during the asthmatic attacks, as a means of active immunization to the causal protein.

Treatment along the lines suggested above for removing or overcoming the cause of the condition are frequently followed by an amelioration of the symptom, and in many instances an absolute cure is effected. Unfortunately, certain cases of bronchial asthma are encountered in which all efforts to detect the causative agent are of no avail, and in which one will have to rely upon such measures as tend to build up the general health of the patient, improve the digestion, assure a favorable climate, and depend upon medication for the relief of the symptoms. Occasionally bronchial asthma may occur in patients suffering from some other disease, such as chronic heart disease, nephritis, tuberculosis, bronchitis, and emphysema, in which case the underlying disease must receive appropriate treatment.

Of all the various drugs recommended in this condition there is none which is of so much value between the attacks in the majority of cases as the iodid of potassium. In 5-, 10-, 15-, or 20-grain doses (0.32, 0.65, 0.97, or 1.3 Gm.) three times a day after meals, preferably administered in essence of pepsin, it will not infrequently result in complete relief of symptoms so long as the drug is continued. Unfortunately, the tendency is for the patients to relapse when the drug is discontinued, as may be necessary from time to time, on account of gastric disturbance, or the evidence of iodism. Lobelia in the form of the tincture (15 minims, or 1.0 mil) may be advantageously combined with the iodide in certain cases. While arsenic has been recommended in the treatment between the attacks, and



may be tried in the event of failure with other lines of treatment, the results are as a rule not very satisfactory, and not to be compared with those following the use of potassium iodide. Atropin may prove of benefit in certain cases, the dose being gradually increased until  $\frac{1}{30}$  grain (0.0021 Gm.) is reached, or the symptoms of dryness of the throat, flushing, or loss of accommodation indicate that the physiologic limit of tolerance has been reached.

The attacks of dyspnea, which are extremely distressing, usually call for active treatment. The chronic sufferer from this disease will soon learn by experience the line of treatment affording the greatest relief, not infrequently instituting certain measures before the arrival of the physician. The breathing is usually much easier with an abundance of fresh air, although care must be taken to protect the patient's body from undue chilling, as the asthmatic sufferer will frequently throw off all covering in the effort to relieve the chest of all restraint. The upright position, with the arms braced to aid the muscular efforts to breathe, is usually assumed by the patient of his own accord.

When it is certain that one is dealing with true spasmodic asthma, there is no remedy which is of so much value as morphin, given hypodermically in combination with atropin. For obvious reasons, however, care should be taken in administering it, and its use should not be instituted lightly, and only when other measures have failed. The relief which follows its use may readily lead to the patient's seeking an injection upon the slightest indication of dyspnea, not infrequently with the development of a habit which leaves him in a worse condition than before. Heroin may be used in its place, or sodium bromid; chloral hydrate, while very effective, in many cases is too depressing to be employed save exceptionally. The sponging of the nose and throat with a 5 per cent. cocain hydrochlorate solution is open to the same objection as morphin, namely, the establishment of a habit, but occasionally it may be employed with benefit in cases of extreme severity which have failed to respond to the other measures suggested.

For many years the nitrites have been held in high repute for the treatment of the paroxysms, and in many cases



deservedly. Nitroglycerin ( $\frac{1}{100}$  grain or 0.00064 Gm.) hypodermically, or amyl nitrite inhalations, may give a great deal of relief when administered early in the attack. The burning of papers saturated with potassium nitrate is another popular method of applying this remedy through the inhalation of the fumes. The smoke obtained by burning certain leaves, such as stramonium, lobelia, belladonna, hyoscyamus, and tobacco combined with powdered potassium nitrate is a very common remedy in this affection, the powder being burned openly in the patient's room, or being used in the form of cigarettes. The use of these powders may occasionally prevent the occurrence of attacks so long as they are used, but the effect soon wears off, no relief being obtained from them after being used for a short time, a statement which is equally true of many of the drugs for the relief of this symptom. A formula for such a powder which has been recommended is:

℞ Stramonii foliorum ..... ʒiv (15.5 Gms.).  
 Anisi fructus,  
 Potassii nitratis ..... āā ʒij (7.77 Gms.).  
 Tabaci foliorum ..... gr. v (0.32 Gm.).

A teaspoonful (4 mils) of the powder may be burned on a plate openly in the room, or covered with a cone through which the smoke is inhaled. Ipecac, apomorphin, and similar drugs in some cases may give considerable relief, especially in patients in whom the dyspnea is accompanied by a hard, dry cough.

Adrenalin has of recent years been very popular as a means of aborting the paroxysms, and while the effect is not very lasting, the relief from the dyspnea is very decided when this animal extract is given hypodermically in doses of 5 to 15 minims (0.32 to 0.97 mil) of the 1:1000 solution in normal salt solution. The injections may be followed by unpleasant symptoms, such as pallor, chilliness, palpitation, and restlessness, but these by-effects are usually transient, and have been considered negligible in view of the relief obtained. While usually considered harmless, adrenalin should be used with extreme caution when there is organic disease of the heart or kidneys, and cases of asthma in which there was cardiac weakness or disease have been reported in which unfavorable

symptoms of a serious nature followed its injection in moderate doses.

While it seems hardly necessary to refer to the use of the various commercial "asthma cures," which depend largely upon the presence of cocain for their effect, it might be well to emphasize the importance of cautioning patients suffering from this disease about the dangers attending their use. Some of the commercial powders for burning in the room contain small amounts of powdered opium, a fact which impresses on the physician the importance of prescribing personally a mixture for burning in the patient's room rather than depend upon the proprietary preparations.

### HAY-FEVER AND HAY-ASTHMA.

The lachrymation and sneezing, so characteristic of hay-fever attacks, may be further aggravated by implication of the bronchial mucous membrane, with coughing and attacks of dyspnea identical in character with the attacks of asthma resulting from animal emanations, food, and similar irritants. The symptoms of hay-fever are so well known and so characteristic that they require no further reference. The condition is so extremely distressing, in spite of its relative innocuousness, that the description of the various methods of treatment are of considerable interest.

The disease is caused by the pollen of various plants, although a certain susceptibility on the part of the individual is necessary for the development of the disease. Bacterial infection and certain deformities of the upper air passages seem to bear an etiologic relation in some cases. While the pollens of various plants are capable of causing the disease, the large proportion of cases arise from the pollen of only a few species, the disease in each individual being apparently due to the pollen of one special plant. The attacks may occur at different periods of the year in different individuals, depending upon the plant life in different climates, but is most common in the late summer (August) and early fall months. When a person with the disease goes to a region where the offending plant does not grow, or when such a plant is eradicated from the neighborhood of the patient, the disease does not occur,

The pollen seems to act in a twofold manner, direct and indirect, in exciting the disease. The first is a local, irritating effect of the spiculated pollen upon the mucous membrane of the nose, while the second excites phenomena of anaphylaxis in sensitized individuals, from the absorption of certain proteins or toxalbumins contained in the pollen, the nasal, ocular, and bronchial symptoms being the evidence of anaphylaxis. This indirect or anaphylactic effect is far the more important and the more common.

In the United States the plants which cause the greatest number of cases are the grasses (timothy, rye, and orchard grass), producing the spring types of the disease, and the rag-weeds, cockleburs, and wormwoods, responsible for the late summer and fall forms. It has been shown that the common ragweed (*Ambrosia artemisiæfolia*) and the giant ragweed (*Ambrosia trifida*) are responsible for 85 per cent. of all cases of autumnal hay-fever in the sections in which these weeds are prevalent. It is of interest to note what a well-organized, intelligent campaign may succeed in accomplishing against a single disease. The number of hay-fever cases in New Orleans has been greatly reduced by a systematic destruction of the hay-fever weeds in the vicinity of that city, the destruction of the weeds being accomplished by education of the public, supported by the necessary legislation.

The general treatment of hay-fever and hay-asthma necessarily includes those measures suggested for the treatment of asthma, namely: correction of nasal defects and deformities; the removal of all possible sources of infection in the nose, throat or mouth; the elimination from the diet of such food-stuffs as may possibly give rise to anaphylactic phenomena (as determined by cutaneous tests), and careful attention to the regulation of the bowels. The appropriate treatment applied to any associated bronchitis, cardiac weakness, or renal insufficiency is also indicated.

The drugs which seem to be of most value are the alkalies, such as sodium bicarbonate, which may be given in 10- to 15-grain (0.65 to 0.87 Gm.) doses every three hours for a few days, and then reduced in amount. If the bicarbonate of sodium should give rise to irritation of the gastric mucous membrane, a small amount of bismuth subnitrate, 3 to 5 grains

(0.19 to 0.32 Gm.), may be added to each dose of the sodium bicarbonate. Calcium salts have proved of considerable value in certain cases, the lactate or chlorid salt being the form in which it is usually employed. These calcium salts should always be well diluted before being administered, and are preferably given after meals. Quinin in large doses and antipyrin have been recommended in the treatment of this disease, but the result of their employment is very likely to be disappointing.

The distressing nasal symptoms may be very much ameliorated by warm alkaline sprays, or by sprays of adrenalin solution (1:10,000), although the use of this latter drug, while very striking at the time, is frequently followed by a period of aggravation of symptoms as soon as its effect has worn off.

When the nasal secretion is excessive, atropin sulphate,  $\frac{1}{300}$  grain (0.00021 Gm.) every two or three hours, until dryness of the throat develops, may prove of considerable value.

In the prevention of the disease the most natural method would be the destruction of the weeds, the pollen of which causes the disease, in the immediate neighborhood of the patient. This is naturally not always possible or advisable, especially in the cases of grass pollen sensitization, and the pollen may be carried by the wind for a considerable distance. If the financial resources of the patient permit, he may be relieved of all symptoms by going several weeks before the expected attacks to some resort free of the weeds causing his hay-fever. Some patients experience considerable relief from an ocean voyage. The most popular of these hay-fever resorts are in the White Mountains of New Hampshire, and in the northern part of Michigan or Wisconsin. There are also several resorts along the New Jersey coast which are regarded as free from this disease. This method of traveling in order to avoid the attack of the disease, while possibly agreeable, is not always convenient, and it is only possible for the wealthy.

*Specific Treatment.* The value of this method of treatment is at present almost entirely confined to its application as a preventive measure, being of very little (if any) value as a curative agent. In rare instances the disease has been aborted in the early stages by means of pollen extracts, but at this period its use is not without danger, not only of aggravating the

symptoms, but of causing serious trouble. Regardless of the specific treatment employed, no measure should be neglected which offers any possible aid in preventing the recurrence of the disease.

The first step in specific treatment is to determine the pollen responsible for the attacks by means of the cutaneous tests described under Asthma. (See p. 352, *et seq.*) Having determined the type of pollen to which the individual is sensitized, various dilutions of this pollen may be employed in a second series of cutaneous tests to decide upon the initial dose to be employed in that individual. The largest amount which fails to excite a positive cutaneous reaction is employed for the first dose, which is not given until all symptoms of local cutaneous reactions have disappeared. The course of treatment should be begun about eight weeks before the attacks usually begin. Ten to 15 injections at three-day intervals are usually required to produce immunity. If symptoms of hay-fever or asthma should develop during the course of the treatment, or any signs of anaphylactic reaction, the subsequent injection should be lower, and the intervals between injections slightly increased. The individual pollens should be employed whenever possible. While the mixed pollens of spring or fall types, as usually supplied by the commercial houses, may be employed, care must be exercised in their administration. The injections of pollen should always be performed cautiously, care being taken to see that the initial dose is not excessive, as an overdose may cause symptoms of an extremely serious nature. It is uncertain how long the immunity persists after such a course of treatment, but it probably does not last for more than two years, and in certain cases it may be quite impossible to produce immunity artificially. Some writers believe that better results are obtained if, in addition to the pollen desensitization, the patient is treated with bacterial vaccines, preferably the autogenous vaccines.

### EMPHYSEMA.

Several different conditions are included under the name of emphysema, the most common being diffuse vesicular emphysema, which is characterized by a permanent overdis-



tension of the air vesicles, with secondary atrophy of the alveolar walls, which is also known as true large-lunged, or hypertrophic emphysema. Senile, atrophic, or small-lunged emphysema occurs in elderly people, and consists of an atrophy of the alveolar walls as part of the general senile atrophy. In both of these types the process is general, involving both lungs equally, although the process may be more marked in certain portions of the lungs. Local or compensatory emphysema, on the contrary, only affects certain portions of the lung or lungs, and is secondary to some other pulmonary disease or lesion. It is most commonly seen in lungs in which a certain portion is crippled by a destructive or contracting process, such as pulmonary tuberculosis, especially when partly or completely arrested, and with cavity formation, or pulmonary fibrosis. The emphysema may occur in the air-bearing lung tissue adjacent to the lesion, or in the opposite lung when the disease causing it has rendered inactive large portions of the lung, as in pleural effusion and pneumonia. This compensatory type should be called simple pulmonary overdistension, as when the causative factor is removed the lung may regain its normal size and condition, although it may eventually result in true emphysema if the overdistension persists for any length of time. There is also an interlobular or interstitial type of emphysema, which is due to an escape of air into the interstitial tissue, and has nothing in common with true emphysema, corresponding more closely to subcutaneous emphysema.

The most important type from a clinical standpoint is the diffuse vesicular emphysema, the others being more particularly of pathologic interest, with the possible exception of the compensatory emphysema.

**Diffuse Vesicular Emphysema.** While some writers recognize this as an idiopathic or essential disease, from a practical standpoint it is so frequently associated with other lesions of the bronchi and lungs, which appear to bear an etiologic relation, that it should be considered as almost invariably a secondary process. The most common cause is chronic bronchitis, either as a result of infection or of cardiac insufficiency. Bronchial asthma comes next in frequency, obstruction or compression of the air-passages being less frequent. That

players upon wind-instruments, glass-blowers, and singers are especially prone to this disease is an observation or theory which has been handed down from one medical generation to another, but later investigations have done much to discredit this view. It is essentially a disease of middle life or old age.

The theories advanced to explain the pathogenesis of emphysema are varied, and, as a rule, unsatisfactory, one group claiming that certain mechanical defects of the chest wall are responsible for the condition, the other group believing that some essential change in the lung itself is the primary cause. Rigidity of the chest wall, as a result of changes in the costal cartilages, is a secondary phenomenon. Changes in the connective tissue, elastic tissue, nerves, or blood-supply have been variously described as the essential conditions upon which emphysema depended. In the light of our present knowledge the most that can be positively stated is that repeated or long-continued distension of the pulmonary tissue may lead to emphysema in certain individuals. Heredity is supposed to be a factor in a certain proportion of the cases.

The pathologic conditions associated with emphysema consist of an enlargement of the chest which is most marked in the anteroposterior diameter, resulting in a barrel-shaped thorax. The costal cartilages in many instances are less elastic and firmer than normal, and even may be enlarged or calcified. The lungs are voluminous and inelastic, the edges are rounded, and may meet in the median line, and usually are of a pale grayish color, relatively dry, and deficient in coloring matter. In advanced cases, immediately beneath the pleura, or deeper in the lung, may be found air vesicles which vary in size from a pin-head to a pea, or rarely even larger. On microscopic examination the alveoli are found to be enlarged with an atrophy of the alveolar walls, and in certain areas small vesicles are seen from the rupture of the walls resulting in the coalescence of numerous alveoli, which may implicate one or more infundibula. Evidence of compression and obliteration of the capillaries is usually present, and the elastic fibres appear reduced in size and number on account of the dilatation of the alveoli. The signs of bronchitis are almost invariably present, and this change may be accompanied by peribronchial induration.

Dyspnea and cough are constant symptoms of emphysema, varying in severity with the nature and extent of the associated conditions, such as bronchitis, cardiac weakness, and the degree of pulmonary distension. The cough and expectoration are in no way peculiar, and merit no special consideration, the subject being considered in detail in the sec-

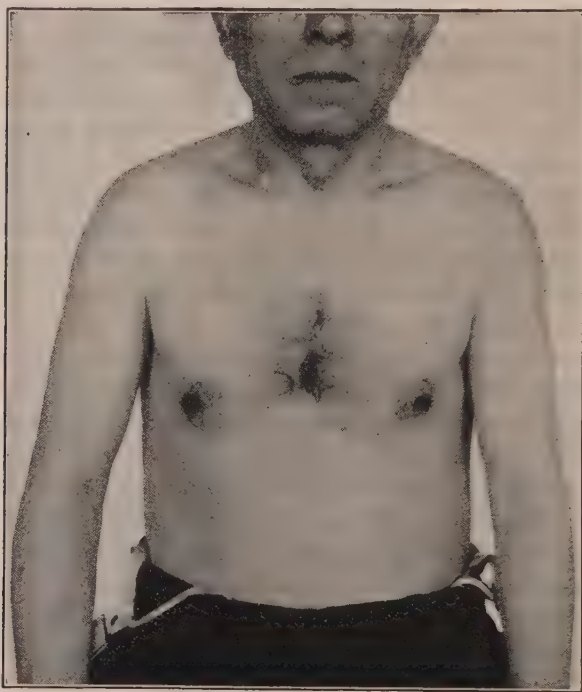


Fig. 3.—Chest changes in chronic emphysema of the large-lunged type.

tions on Bronchitis (*q.v.*). The dyspnea may only be present upon exertion, the breathing being apparently normal when the patient is at rest, or it may be constant and severe. In certain cases the dyspnea may be paroxysmal, simulating bronchial asthma, or the asthma-like attacks which occur in chronic cardiac disease.

The diagnosis of emphysema may be made upon inspection alone in many instances, especially when the disease is of long standing. Cyanosis is almost always present, and, while

usually slight, may be very severe. Dilatation of the superficial veins is frequently present. The respiratory movements are labored, the accessory muscles of respiration being visibly brought into play. The chest is large and appears to be in a constant state of expansion, respiratory movements of the chest being more in the nature of an up-and-down motion of



Fig. 4.—Same patient as in Fig. 3. Note the increased antero-posterior diameter of the chest.

the anterior portion of thorax, the normal range of costal movement being absent or diminished.

The antero-posterior diameter of the chest is markedly increased in relation to the lateral enlargement, giving it a cylindrical or barrel-shaped appearance on cross section, in contrast to the normal oval or kidney-shaped outline. This may be clearly seen if a tracing is made of the outline of the chest by means of the cyrtometer. The short, thick neck,

shoulders high and slightly stooped, prominent sterno-cleido-mastoid muscles, the obtuse epigastric costal angle, the enlarged barrel-shaped chest, and the labored breathing and diminished respiratory excursion of the chest walls together form a picture which is absolutely characteristic in the majority of cases. Palpation reveals diminished tactile fremitus, and the apex beat of the heart is very feeble, or cannot be felt at all. On percussion, the sound elicited is booming, loud, and low-pitched—hyperresonance, the resonance extending several interspaces below the normal lower limits, or the lower border

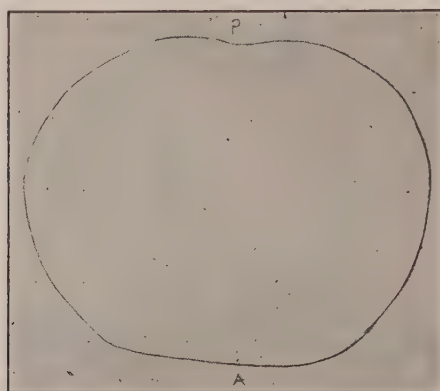


Fig. 5.—Chest tracing of patient shown in Figs. 3 and 4. Note the circular outline, with increased antero-posterior diameter, which is characteristic of emphysema.

of resonance is more easily determined, indicating that the diaphragm is less convex than normal, and that the pulmonary lower edge is more voluminous and thicker than in the healthy lungs. The area of so-called absolute cardiac dullness (cardiac flatness) may be diminished or absent, resonance being obtained over the entire precordia. The inspiratory excursion, as measured by the movement of the lower border of resonance, is distinctly diminished. The upper borders of hepatic and splenic dullness are lower than normal, and more difficult to map out. Auscultation reveals a prolonged, soft, low-pitched expiratory murmur, when not obscured by the sonorous or sibilant râles, which are usually of prolonged duration, al-



though the râles may be at times distinctly moist and of varying size.

The various changes in the heart as a result of increased resistance in the pulmonary circuit can usually be determined, especially in the right heart. The *x*-ray may be necessary to determine the exact size of the heart, and may be used to confirm the physical findings. The diminished expansion also may be further studied by means of chest measurements or with the spirometer.

### TREATMENT.

The treatment of true emphysema consists mainly in the control of such secondary conditions as may aggravate or be responsible for the symptoms, no means in our possession at the present time being capable of actually overcoming the overdistension of the alveoli and renewing their contractility.

The dyspnea may be relieved in certain cases by treatment directed toward correcting abnormalities of the circulatory organs or relieving diseased kidneys. In gouty or rheumatic subjects some relief may be obtained by treatment of the underlying systemic disease. There is no condition outside of the bronchial catarrh which seems to aggravate this symptom to so marked a degree as disturbances of digestion, especially when associated with gaseous fermentation in either the stomach or the intestines. Careful attention to diet, and the correction of any tendency to constipation, with such medication as may be indicated to correct the disturbances of the gastro-intestinal tract, will in certain cases be invaluable in the relief of the symptoms of this condition.

The commonly associated bronchitis is usually very resistant to treatment, and is the symptom toward the relief of which most of the medication will have to be directed. In the majority of cases, the degree of comfort which the sufferers from this disease enjoy is in direct proportion to the extent to which the bronchitis may be controlled. The section on the treatment of Bronchitis (*v.s.*) could be inserted under this section with perfect propriety. The bronchitis may be spasmodic in character, simulating an attack of asthma, in which event such remedies as lobelia, belladonna, and potassium iodid may prove of benefit. Morphin in such cases

should be employed with the greatest caution, and never in cases accompanied by evidence of marked bronchitis or bronchiolitis. When there is considerable wheezing, usually occurring in spells, relief may be obtained by counterirritation of the chest with mustard, iodin, or turpentine, and by the administration internally of potassium iodid.

When cyanosis is present, digitalis and active purgation may prove of benefit, and in rare, severe cases, venesection may become necessary. The general condition of the patient should be very carefully watched, and every measure resorted to which will tend to improve it. The questions of clothing, bathing, sleeping, and dietary should be made the subject of a detailed study, and the patient instructed in regard to the amount and character of the exercise, and warned of the necessity of avoiding overexertion and fatigue.

Where circumstances permit, the patient should live in that climate which seems to relieve him of his symptoms and provide the greatest amount of comfort, the selection of the climate being dependent upon personal idiosyncrasies for which we have no fixed rules for guidance. It will usually be the climate in which the patient is least likely to be affected with attacks of bronchitis.

Various mechanical devices, such as pneumatic cabinets of various kinds, have been recommended in the treatment of emphysema, and while considerable ingenuity has been displayed in their construction, the results obtained from their employment have not been sufficiently successful to warrant their being generally employed. Breathing exercises, massage, forcible compression of the chest, and similar measures may be employed with occasional benefit, especially in the earlier stages of the disease.

Emphysema is extremely resistant to treatment, and the best that can be expected is an amelioration of the symptoms. As previously stated, the medication employed must be directed toward the correction of the abnormalities present in the other organs of the body, or the relief of the associated conditions which aggravate the symptoms. In some cases strychnin, atropin, and caffein may be administered with some benefit, in addition to the other lines of treatment suggested. Adrenalin has been recommended in those cases in

which the cough is of a spasmodic character, and where persistent wheezing is so marked as to constitute a very distressing symptom. The employment of the drugs just mentioned must necessarily be undertaken with a certain amount of caution, and only where no contraindication exists.

It is too soon to draw conclusions as to the utility of surgical interference, based on Freund's theory of costal cartilage changes. The uncertainty existing in regard to the theory upon which these measures are based should make one extremely doubtful of the value of the surgical measures suggested.

**Compensatory Emphysema.** The pathologic process is the same as in the type just described, with the exception that only portions of one or both lungs, or one entire lung, is affected instead of a generalized lesion of both lungs. The condition may not be demonstrable clinically, but when so marked as to be recognizable the physical signs are the same as those of diffuse vesicular emphysema. The process attains its greatest clinical interest in those cases of pleural effusion, pulmonary tuberculosis, or pulmonary fibrosis, in which the unaffected lung may become markedly overdistended, on account of loss of function of the greater part of one lung. The one-sided pulmonary dilatation may interfere with the usual physical findings in such conditions, and may tend to mask deep-seated processes developing in the emphysematous lung.

This compensatory type of emphysema is in reality a functional hypertrophy, or at least, it should be looked upon as such a condition, and hence it requires no special treatment other than that directed toward the process which has caused its development. When the dilatation has persisted for some time, the condition may become permanent, giving rise to the symptoms associated with the diffuse vesicular type, in which case the same line of treatment should be pursued as suggested for the latter condition.

**Senile Emphysema.** The senile, or atrophic, form of emphysema is an evidence of the wasting which takes place in the aged, the lung sharing in the general atrophic or involution process affecting the entire body. The elasticity of the lungs is diminished, the chest assuming a position approaching

that of permanent inspiration. The lungs are smaller than normal, in distinct contrast to the other forms of emphysema above described.

The symptoms are not nearly so marked as in the diffuse vesicular form. There may be little or no dyspnea present, owing to the limited capacity for physical exertion due to the general muscular weakness. Where marked bronchitis is associated with the process, spasmodic attacks of severe dyspnea may occur. The patients present a general atrophic, withered appearance, without the evidences of venous obstruction so commonly seen in the hypertrophic type. The chest

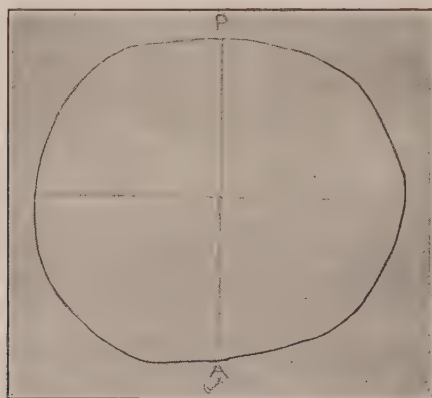


Fig. 6.—Chest tracing in senile emphysema of the atrophic or small-lunged type. The retraction on the left side anteriorly is due to an old fibroid process.

presents a rounded appearance, not as a result of increase in the antéro-posterior diameter, but as a result of a general shrinkage which mainly affects the lateral diameter. The ribs assume a more oblique direction, the interspaces over the lower portion of the chest being narrowed or even obliterated. The signs are similar to those found in diffuse vesicular emphysema, except that there is no evidence of enlargement of the lungs, and the expiration is not prolonged to the same extent.

The senile, small-lunged type of emphysema should be treated as one would treat the diffuse, large-lunged type, and the prospect of improvement in the senile type is even worse than in the latter form.

## PULMONARY TUBERCULOSIS.

The tubercle is the basis of practically all pathologic processes resulting from an implantation of tubercle bacilli in tissues favorable to their growth. Microscopically, the tubercles in the early stages of their development present a characteristic appearance, the center being occupied by giant cells and epitheloid cells surrounded by a zone of concentrically arranged epitheloid cells, around which is arranged a zone of round cell infiltration composed principally of lymphocytes. The subsequent changes in the tubercle, whether caseation or conversion of the tubercle into fibrous scar tissue occur, are of considerable clinical interest. Even when caseation takes place in a tubercle, it may become encapsulated by fibrous connective tissue, the contents undergoing softening and evacuation, or becoming inspissated, and possibly calcified.

The extent to which either of these processes, caseation or sclerosis, predominates in the individual case of pulmonary tuberculosis largely determines the clinical course of the disease.

The first foci of pulmonary tuberculosis usually are found in the region of the apex of the lung, at a point slightly below (about  $1\frac{1}{2}$  inches [3.81 cm.]) the extreme apex, and usually nearer to the posterior than to the anterior border of the lung. The process may be limited to this region or it may extend until the entire upper lobe is gradually infiltrated. As the process extends downward the lower lobe may become affected, and at this time it is extremely common to find evidence of some infiltration in the other lung, either at the apex or at the root.

The appearance of the lesion varies greatly in different cases, depending upon whether the destructive or reparative process predominates, and upon the course taken by the disease in its extension. There may be numerous minute tubercles or large conglomerate tubercles, the disease progressing by the direct enlargement of the tubercle, and by the development of fresh deposits in its immediate neighborhood. The portion of lung invaded may be merely the peribronchial tissues or the alveolar parenchyma. A large portion of the lung tissue may be affected by a caseous pneumonic process, or the tubercles may be disseminated, with air-bearing tissue remaining between them. The recent tuberculous areas are occasionally found surrounded by a



zone of congestion, which varies greatly in extent. More frequently a certain amount of sclerosis surrounds the tuberculous areas, limiting their growth by extension. The amount of fibrous tissue is extreme in some cases, thick bands of scar-like tissue surrounding the tuberculous areas and extending into the mass, or with fibrinous striæ running out into the lung. The sclerosis in some cases may be the only evidence of the disease, where the fibrosis has followed closely upon each fresh extension of the process.

Where large areas of the lung tissue have been crippled by tuberculosis of the proliferative type, extensive patches of caseation may be seen, in which softening may or may not be present. Softening and liquefaction of the areas of caseous degeneration lead to the formation of cavities, the walls of which are usually formed by fibrous tissue. It is uncommon to find extensive areas of softening and liquefaction, with the formation of very large cavities, in rapidly progressing cases, numerous small cavities being more commonly present in this group of lesions. Cavity formation usually takes place when areas of dense infiltration have been walled off from the rest of the lung by scar tissue.

The healing of pulmonary tuberculosis consists of the formation of fibrous tissue about the tubercles, and when the tubercles are small the result may be a complete conversion into scar tissue, no evidence of tuberculosis remaining. Large tubercles in which caseation has occurred may be walled off, inspissated, and later calcified. When the tubercles are very large they may also become walled off, the contents undergoing liquefaction, and persisting as a closed abscess cavity, or it may rupture into a bronchus with the evacuation of its contents through that channel. Cavity formation should be looked upon as a healing or reparative process in probably the majority of instances. This is especially true of the small cavities occurring at the apex, with the evidence of only a slight amount of additional implication of the lung. While bronchiectatic cavities may occur in tuberculosis, they are not common, being most frequently seen in the fibroid types of the disease. Extreme cavitation of the entire lobe, or even one entire lung, occasionally may be present.

**Tuberculous Resistance.** The character of the disease process which develops as a result of an implantation of tubercle bacilli varies greatly in different individuals, ages, and races. The

conditions which influence the nature of this tissue response to the tubercle bacilli do not depend upon the virulence of the invading micro-organisms, as this has been shown to be almost constant. The accidental location of the infection plays a part in determining the resulting process, not only the organ which it affects, but also the relation of the tubercle to blood-vessels and lymphatic channels, and possibly the severity of the infection, as indicated by the number of invading bacteria, and whether it represents a primary infection or not. The most important factor seems to be the property possessed by the tissues to react to the presence of the invading micro-organisms in various ways, which has been called resistance, for lack of more definite knowledge of the mechanism of the process. The degree of resistance to tubercle bacilli varies to a marked extent in different individuals, resulting in a process which ranges from the rapidly progressing form of tuberculous disease to the type in which the infection does not result in any actual disease at all; the tubercle bacilli being completely walled off by connective tissue from the surrounding tissues. This complete walling off of the invading bacilli constitutes the nearest approach to immunity that has been demonstrated in man. The immunity is therefore at its best only relative, being an immunity to the development of disease, but not to infection. Even in those cases in which the primary implantation is walled off from surrounding tissue the bacilli may remain dormant for years, retaining their viability and pathogenicity, and it is possible for these bacteria to develop and to set up a morbid process whenever conditions favorable to such development arise. Upon this peculiar ability to lie dormant for years is based the theory that all primary infections occur in early childhood, the disease arising in later life being viewed as a transformation of the early infection into an active process, or possibly in some cases as the result of a secondary infection, the theory being that the primary infection rarely is immediately followed by tuberculosis, nearly every individual possessing the ability to wall off the first implantation. The subject is one that involves many different factors, and many questions remain which are far from being positively settled. It is unfortunate that we have no means at the present time of accurately determining the relative resistance in the individual case, as upon this fact depends not only the question as to whether infection alone or tuberculous dis-

ease results, but also the general character of the disease process. Some writers believe that the development of pulmonary tuberculosis is determined by the presence of certain anatomic changes in, or abnormalities of, the chest wall. The ossification of the costal cartilage and the shortening of the ribs lead to stenosis of the bony thorax, the first rib being especially affected. The theory is plausible, and while it does not fully explain the tendency to the development of tuberculosis of all cases, these deformities of the thorax seem to play a very important rôle in many tuberculous subjects.

From the clinical standpoint, it is extremely important to bear in mind that it is possible to have an infection without definite tuberculous disease, and it is of equal moment to be able to distinguish between the two conditions. Upon this ability to make a distinction between infection and disease will depend the accuracy of the prognosis and the necessity for treatment, and upon the ability to estimate the degree of resistance in the individual instance, as evidenced by the type and character of the tuberculous process, will depend the ability to institute the proper method of treatment. While what has been said applies especially to pulmonary tuberculosis, it is equally true of the disease when implanted in other parts of the body.

*Von Pirquet's Test.* Infection with tubercle bacilli does not always result in tuberculous disease, probably the majority of infections giving no evidence of their presence during the life of the infected individual. The presence of such infections may be determined by the Von Pirquet cutaneous tuberculin test, applied in the following manner: The skin of the arm or forearm is cleansed and dried with a piece of sterile gauze. By means of a Von Pirquet borer the superficial layers of the epidermis are removed at three points about 1 to 2 inches (2.54 to 5.08 cm.) apart, in a line parallel to the long axis of the arm. The central point is allowed to remain undisturbed, to serve as a control. A small quantity of Koch's old tuberculin is applied to the distal and proximal points by means of a glass rod, and gently rubbed into the skin. After allowing the tuberculin to remain in contact with the skin for five or ten minutes, the surplus is removed and the surface allowed to dry. After the tuberculin has become sufficiently dried by exposure to the air, the points

of inoculation are covered with a piece of sterile gauze, held in position by narrow adhesive straps, which are not permitted completely to encircle the arm. The gauze should be removed at the end of twenty-four hours, and the inoculated points inspected. If no reaction is evident, the examination should be repeated at the end of another twenty-four hours. A positive reaction shows a distinct redness and elevation in the region of the points at which the tuberculin has been applied, in contrast to the control point. The reaction may appear within a few hours after the test has been applied, but usually persists for a sufficient time to be evident at the end of twenty-four hours. Frequent inspection after the test has been applied is probably safer, but is not always practicable. The induration in and about the inoculated points may persist for several days. If the first application of the test is negative, it may be repeated at the end of a week, or even a third test may be necessary. A positive reaction merely indicates that the individual has at some time been infected with tuberculosis, and this should never be interpreted as an indication that active tuberculous disease is present. The determination of actual tuberculous disease is much more difficult, and demands a careful inquiry into the patient's previous history, a minute, detailed record of the symptoms, and a thorough examination of the suspect, including such laboratory and clinical aids as may be of value.

The diagnosis of tuberculosis by means of complement fixation tests has been receiving considerable attention in recent years. The method has not been applied to a sufficient number of cases to warrant one in drawing positive conclusions from the results obtained, as to whether it will ultimately prove of value in the diagnosis or prognosis of tuberculosis. It is to be hoped that further investigation and observation will result in providing a method of examining the blood which will be of value in the diagnosis of the presence of tuberculous disease, and will supply a means of estimating the degree of activity of the process.

Too much emphasis cannot be laid upon the importance of determining which cases are in need of radical treatment owing to the presence of actual disease. The diagnosis of tuberculosis conveys to the patient's mind a picture of "con-



sumption." with all the misery, pain, and discomfort which that name suggests. The very word tuberculosis implies to most people a breaking up of their home and a rearrangement of their entire life, with usually a serious economic sacrifice. With the prevalent views in regard to the contagiousness of the disease, the diagnosis also entails the placing of a certain stigma upon the person affected, from the effect of which he may never fully recover. It is extremely important, therefore, that every care should be used in making a diagnosis of tuberculosis to avoid placing such a burden upon an individual unnecessarily. It is equally important that the diagnosis of tuberculosis should be made in every individual suffering from tuberculous disease at a time when the lesion is slight, before the lung has been very much damaged, in order that the proper line of treatment may be instituted during a period when his chances for recovery are at their best, so that it may be possible to restore him to full working capacity. Having made a diagnosis of tuberculous disease, it is imperative that the patient be informed of the fact, if one expects to receive the co-operation of the patient in carrying out the necessary remedial and preventive measures. As the hope of recovery can be held out to the majority of patients, there is no reason why the patient should not be informed of the nature of the disease from which he is suffering, except possibly in rare instances where extraordinary circumstances may exist.

In taking a history of the family or of previous diseases it is advisable to accept no statement of a patient on faith, every effort being made to substantiate or disprove every statement by careful cross-examination. This is especially important in regard to the disease which may have caused the death of any member of his family, or from which he may have suffered. By having him describe the symptoms of the illnesses in detail, it is frequently a simple matter to show that the name given by the patient to the disease is misleading, if not erroneous.

The previous history of the patient should not be confined to attacks of illness, but should include an inquiry into the general health and strength of the individual, dating back to early childhood whenever possible, and whether they have been subject to such minor (?) complaints as colds, la grippe,



bronchitis, indigestion, and similar maladies. In determining the mode of onset of the present illness, the previous health and symptomatology of the individual the greatest care must be exercised, as upon the information obtained alone, in the large majority of instances, it is possible to make a diagnosis of tuberculous disease, the physical examination being merely necessary to confirm the diagnosis, localize the disease, and to determine the extent and character of the lesions. The physical examination is mainly of value from the standpoint of prognosis and treatment, rather than diagnosis, in a large proportion of cases. Many practically healthy persons present physical signs of some abnormality at an apex of the lung, but no one can suffer from active tuberculosis of the lungs without symptoms, even if in certain cases it may be impossible to locate the focus of disease. It must be apparent why it is necessary to go into the question of symptoms in the most thorough manner, especially as it requires no special training, such as physical diagnosis demands.

The mode of onset varies greatly in different cases. In some the symptoms referable to the respiratory tract predominate; in others the symptoms are in a general way merely suggestive of a chronic toxemia. The various clinical pictures of the early stages of the tuberculous disease may be easily formulated, when one realizes that it is usually diagnosed "typhoid pneumonia," typhoid fever, malaria, chronic bronchitis, chronic gastritis, neurasthenia, anemia, and chlorosis. The only type which should cause any confusion is that in which the earliest and most prominent symptoms are referable to the gastro-intestinal tract, since in these examples of the disease the symptoms which should direct attention to the possible presence of pulmonary tuberculosis may be relatively slight.

The various symptoms which may suggest the presence of pulmonary tuberculosis will be considered under separate headings. It is to be regretted that space will not permit their receiving that detailed consideration their importance deserves.

*Cough and Expectoration.* Cough is the most common symptom of phthisis, and in the majority of cases the one which first attracts the attention of the patient. The cough may be transient, occurring only during the winter months, or it may persist for a long period of time; it is usually worse at night on retiring, or on

rising in the morning, and in some patients the attacks occur after eating. There is no characteristic of the cough which is at all peculiar to pulmonary tuberculosis; it may merely be a mild hacking cough, or may be extremely severe and exhausting, and the spells may occur in paroxysms, or may be followed by vomiting. This "emetic cough," as it has been called, is believed to have considerable significance as an indication of the presence of pulmonary tuberculosis, in those cases in which pertussis can be ruled out. The cough is usually accompanied by the expectoration of mucopurulent material in anyone in which the disease is of any prolonged duration or considerable extent. In some the expectoration is not noticeable, on account of its being unconsciously swallowed, or the patient is unable to expectorate the material coughed up. This is especially true in children, where it is frequently impossible to secure material for an examination. In some cases the expectoration is brought up and expelled without any apparent cough accompanying the process. The amount and character of the expectoration varies with the character and location of the pulmonary process, and the extent to which bronchitis accompanies the tuberculous disease.

While the amount of cough and expectoration usually bear a definite relation to the activity and extent of the pulmonary disease, this is not always true, being apparently dependent upon the location of the lesion in regard to the bronchi and the character of the process. In cases in which the signs and symptoms point toward an arrest of the tuberculous disease, the cough and expectoration may persist. This may be due to the presence of a cavity, or to the fact that the fibrosis resulting from the healing has led to deformities in the bronchi, or to an interference with their blood-supply, resulting in a chronic hyperemia or inflammation of the bronchial mucosa.

*Sputum.* The microscopic examination of the sputum may supply information of the greatest diagnostic significance. The most important constituents of the sputum, to be sought for are the tubercle bacilli, which are most conveniently demonstrated by the Ziehl-Neelsen-Gabbett method. When the micro-organisms cannot be demonstrated by the ordinary technic, it is advisable to employ the antiformin method of dissolving and centrifugating the sputum, for the tubercle bacilli may frequently be demonstrated by this method. It has the advantage of concentrating the

tubercle bacilli, so as to permit of examining a large quantity of sputum on one or two slides. When it is imperative that a positive diagnosis be made, it may be necessary to resort to inoculation of the sputum into guinea-pigs, a procedure which has certain disadvantages, but makes the diagnosis absolutely certain when the findings are positive.

The finding of tubercle bacilli in the sputum makes a diagnosis of tuberculosis unquestionable, but unfortunately the absence of tubercles does not exclude such a diagnosis. A negative sputum examination has practically no significance, in so far as excluding tuberculosis is concerned. Even repeated negative examinations must not be accepted as conclusive evidence that tuberculosis is not present. In many cases, even where the disease is of considerable extent, tubercle bacilli have been demonstrated in the sputum only after repeated examinations, and in a small group of cases in which the diagnosis of tuberculosis is fully justified by the clinical findings, it may be impossible to detect tubercle bacilli in the sputum. In the presence of symptoms and signs indicating the presence of pulmonary tuberculosis one should never delay making a positive diagnosis on account of the absence of tubercle bacilli. When they can be identified the case is no longer an incipient one, and the chances for recovery are commensurately minimized. Many cases have lost their opportunity of getting well through the disinclination of their physician to make a positive diagnosis until tubercle bacilli could be found.

Elastic fibres may be found in the sputum in a large proportion of tuberculous cases, and while they do not possess the same diagnostic value they held previous to the discovery of the tubercle bacillus, their presence is of considerable value in doubtful cases (see Pulmonary Abscess). The albumin reaction occurs in the sputum in numerous diseases, and, therefore, its main value lies in excluding tuberculosis when repeated tests have proven negative.

*Fever.* In the early diagnosis of tuberculosis a careful record of the temperature (see Rest and Exercise) is of the greatest possible help. The temperature in children is so variable that much weight cannot be attached to daily variations of considerable extent, but in adults an afternoon rise to 99° F. (37.4° C.) or more must be considered extremely suggestive, in the absence of any obvious cause for such an elevation. A frankly subnormal

temperature in the morning on rising is frequently associated with the afternoon rise, which gives a daily variation of considerable extent. In cases presenting physical signs indicative of apical infiltration of the lung a temperature record of a week or two will frequently be of value in determining the activity of the process, and should always be insisted upon in doubtful cases. Apparently the tuberculous individual is extremely susceptible to factors responsible for an elevation of temperature in the healthy, such as exercise, menstruation, nervous excitement, and so forth. The temperature attributed to such conditions in the tuberculous is usually higher and more prolonged than in the well, in whom it is usually slight and evanescent. When moderate exercise induces a distinct rise of temperature, persisting at the end of one hour's rest, or when in a female there is a decided premenstrual or menstrual fever, tuberculosis should be strongly suspected. When accompanied by other symptoms which point toward pulmonary tuberculosis one would be warranted in making a positive diagnosis, even in the absence of definite physical signs, when no other cause for the rise of temperature could be detected.

*Night-sweats.* Night-sweats may occur early in the disease, although, as a rule, they are more frequently encountered and more severe in the advanced stages. For many years they have been considered as pathognomonic, and while they undoubtedly occur in conditions other than tuberculosis, their presence in association with other symptoms of tuberculosis possesses considerable value.

*Hoarseness.* Hoarseness provoked by changes in the weather, or by prolonged use of the voice, is frequently an early symptom. Even a constant severe hoarseness may be present in cases in which there is no actual laryngeal tuberculosis. Temporary attacks of hoarseness may follow hard coughing spells, being apparently due to the adhesion of small particles of mucus to the larynx. When this symptom is present a careful study of the larynx should be made, in order that the presence of actual disease or of paralysis may be detected in the early stages.

*Hemoptysis.* There is no symptom which is so distressing to the patient, and, when carefully studied, is of so much value in the diagnosis of tuberculosis, as the expectoration of blood. When it can be definitely determined that the blood is coming from the lung, the case should be considered one of pulmonary



tuberculosis until it can be proved that some other cause of hemoptysis exists. In the event of a frank expectoration of blood, too much care cannot be employed in ruling out hematemesis and epistaxis. Hemorrhages from the throat are extremely rare, and when they occur the source of the bleeding may be detected by a careful examination. Among the causes of pulmonary hemorrhage by far the most common is pulmonary tuberculosis, other factors being cardiac disease, bronchiectasis, syphilis of the lung, malignant disease, gangrene, trauma, and vascular hypertension.

While blood-streaked sputum is a frequent sign of tuberculosis, too much weight should not be attached to its occurrence, as it frequently occurs in other conditions. Many of the hemorrhages in tuberculous women occur at the menstrual period, and for this reason are usually called vicarious menstruation. While admitting the possibility that vicarious menstruation may be manifested in the form of a pulmonary hemorrhage in an otherwise healthy woman, any hemoptysis occurring at the menstrual periods should always be viewed with extreme suspicion.

*Gastro-intestinal Symptoms.* Symptoms referable to the digestive tract are extremely common in early cases of pulmonary tuberculosis, preceding all other symptoms in many instances. The appetite is variable, and, while it may be retained, the majority of patients show some disinclination for food. The degree of anorexia seems to bear no relation to the height of the fever, some phthisics retaining their appetite in the face of considerable elevation of temperature. An aversion to fats has been noted among the tuberculous by several observers, and at times the carbohydrates, especially the saccharins, are the foodstuffs to which an aversion arises. The gastric disturbances seen in early cases are in no way characteristic, the symptoms being the same as those associated with a moderate lack of gastric motility and diminished secretion due to any other cause.

*Loss of Weight.* Loss of weight in the early stages of tuberculosis is almost invariable, being frequently the symptom which has first attracted the attention of the patient to the possibility of this disease. The loss of weight is not attributable solely to the digestive disturbances, since it may occur in those whose digestion is unimpaired. Progressive loss of weight in anyone should suggest the possibility of tuberculosis, especially when associated with other symptoms pointing in the same direction. Cases are



encountered in which the subject is not emaciated, but, on the contrary, extremely well nourished, and the presence of a thick deposit of subcutaneous fat does not exclude pulmonary tuberculosis.

*Cardiovascular System.* Cardiac palpitation, tachycardia, and hypotension are the most important functional disturbances of the cardio-vascular system encountered in pulmonary tuberculosis. The attacks of cardiac palpitation may follow upon the slightest exertion or excitement, and occasionally are extremely severe and distressing. Rapidity of the cardiac action is almost constant in all stages of the disease, and may possess considerable diagnostic importance. While a rapid pulse usually accompanies the febrile manifestations of pulmonary tuberculosis, this evidence of toxemia may precede any obvious elevation of temperature, and may persist after the temperature has become normal. The study of the pulse-rate in tuberculosis is important, not only from the standpoint of diagnosis, but because it is also a valuable guide in the treatment of the disease. In a few instances an abnormally slow pulse has been observed in this disease, but the occurrence of bradycardia must be so rare as to be practically negligible.

A certain proportion of those suffering from pulmonary tuberculosis has been repeatedly shown to have a heart of smaller size than normal, whether due to hypoplasia or to atrophy and degeneration of the cardiac substance. Certain writers have attempted to prove that the small heart bears a predisposing relation to the pulmonary disease, but it is doubtful whether the small size of the heart can be considered as directly predisposing to tuberculosis of the lungs,

The blood-pressure is almost always low in individuals suffering from tuberculosis, this peculiarity being so universally true that the presence of arterial hypotension should always suggest the probability of this diagnosis.

*Anemia.* The majority of patients present the appearance of anemia, but this suggestion is not always borne out by the examination of the blood. A diminution in the number of erythrocytes is uncommon, although the presence of a chloroanemia is relatively frequent, the blood-picture being characterized by a distinct and predominant loss of hemoglobin. In the moderately advanced cases it is not unusual to find a polycythemia, with a low color index. The leucocyte changes are hardly constant enough to be of value, from the standpoint of diagnosis.

*Nervous Phenomena.* The vaguely defined group of symptoms termed neurasthenia are frequently encountered even in the early stages of pulmonary tuberculosis. Various reflex nervous phenomena are not uncommon, and such symptoms result from disturbances of the sympathetic system, and are manifested chiefly by localized flushing and sweating and dilatation of the pupils. Pains in the chest are frequently present, but they seem to bear no definite relation to the location or character of the pulmonary lesion, and are reflex in origin in the majority of cases.

*Physical Signs.* From the history and symptomatology in probably a large proportion of cases it is possible to reach a fairly reliable conclusion as to the probable existence of pulmonary tuberculosis in the individual case, the physical examination of the patient being employed merely as a means of confirming such an opinion. In the well-marked or moderately advanced examples of the infection, the facial expression and the general appearance of the suspect may unconsciously influence the examining physician in formulating his tentative diagnosis. In arriving at a positive diagnosis, giving a prognosis, or outlining treatment, it is essential that a physical examination of the patient be carefully made. It is not the intention to describe in detail all the physical signs found in pulmonary tuberculosis, but merely to call attention to the signs which seem to be of most value or are usually neglected. Before taking up the various signs, and the methods by which they may be elicited, it may be worth while calling attention to the fact that there are no pathognomonic signs of pulmonary tuberculosis. For a diagnosis, then, it is merely necessary to discover evidence of disease of the lung, usually in the form of an infiltration, which is confined to or is most marked at the apex of one or both lungs, and occurring in a person who presents the appearance and symptoms of tuberculosis.

*Inspection and palpation* of the thorax are indispensable details of a complete physical examination. There are certain facial characteristics which are extremely suggestive of pulmonary tuberculosis, especially when it is of long standing, but it should be borne in mind that this affection may occur in those who present the picture of perfect health. Atrophy of the facial muscles with prominent malar bones, pallid lips, flushed cheeks, the long thin neck, and stooped shoulders,

together form a characteristic picture of the confirmed consumptive. The appearance of the eye is probably the most striking and typical feature of the face, this change being due to the dilated pupil with the pearly white sclerotic, giving it a peculiar transparent brilliancy, accentuated by being deeply set in the socket. The hands also frequently show characteristic changes, the skin being dusky or pale, and the nails frequently curved, with or without clubbing of the ends of the fingers.

Inspection of the chest, which should always be made with at least the upper half of the thorax exposed, usually gives information of enormous value. Men should be stripped to the waist for a satisfactory examination; and too much emphasis cannot be laid upon the importance of removing the clothing when examining the chest. The exposure is objectionable to women, and can be avoided, without seriously interfering with the examination, by having them remove the corsets and lower the clothing to the waist-line, the chest being covered by a light flannel shawl or a thin kimono-shaped jacket fastened in front and back with a few buttons or tapes. By having the jacket large and full, it may be slipped down in front or in back as desired, only that portion of the chest which is under examination being exposed at the time.

Pityriasis versicolor of the skin of the chest, and dilated venules or enlarged veins below the clavicles, posteriorly in the region of the upper thoracic spines, or along the lower costal margin, are frequently met with in phthisis. A great deal of stress has been laid upon the significance of the phthisical chest, but pulmonary tuberculosis may occur in chests of any type, shape, or size. It is a mistake to attach too much importance to the general configuration of the chest in its relation to the diagnosis of this disease. Much more important is the detection of differences between the two sides of the chest, whether of contour or mobility. Depressions above and below the clavicles are of considerable importance, especially when more marked on one side or the other. The flattening of the ribs over one apex or the other may frequently be visible, but this is always better elicited by the hand placed over the upper chest with the fingers pointing toward the coracoid process.

The flattening of the ribs and the loss of the natural anterior curve can be easily felt with the hand in this position, usually confined to one apex. Diminished expansion over the upper portion of one lung may be observed, or, more commonly, the upper chest on the affected side is seen to lag behind the other, even when the expansion of both sides is equal. This is seen to better advantage by standing in back



Fig. 7.—Pulmonary tuberculosis (right-sided). Note the long, narrow chest, and the marked depression above and below the right clavicle.

of the patient, but it can be elicited best by palpation, especially when it is possible to seize the upper portions of both halves of the chest between the fingers and thumbs. Standing behind the patient with the thumbs posteriorly along the upper and inner edge of the scapulæ, the elbows elevated, and the palmar surface of the fingers pressed against the upper portion of the anterior chest, it is much easier to detect slight

differences of expansion on the two sides, or lagging at one apex on the other. This lagging is frequently present very early in the disease and is of special value in unilateral cases. The flattening and lack of expansion at one apex practically always indicates either long-standing fibroid change or extensive infiltration and considerable loss of tissue from cavity formation. When the symptoms and signs suggest an early lesion in a subject with signs of retraction and loss of expan-



Fig. 8.—Illustrating method of palpating apices to determine their relative expansibility.

sion at one apex, it may be taken as evidence that the present illness is a reinfection or an acute exacerbation of an old fibroid process. Differences in the expansibility of the lower portions of the chest can be best brought out by standing in back of the patient and grasping the chest between the fingers and thumbs, with the thumbs posteriorly, by comparing the excursion of the thumbs in their relation to the spine (which may be marked with a blue pencil) during forced respiration. By this procedure slight differences can



be detected readily, if care be used to make equal pressure on the two sides of the chest. The hands should rest as lightly as possible against the chest wall, undue pressure being avoided, as only sufficient pressure is needed to maintain the



Fig. 9.—Method of palpating lower portion of chest to determine relative expansibility of the two sides.

hands in close apposition to the walls of the chest. The difference in expansion of the lower chest may be due to pleurisy, or to tuberculous invasion of that portion of the lung on one or the other side.

In addition to the above technical methods, inspection and palpation should be employed to locate the apex beat and to

detect abnormalities in the chest or abdomen which might have any possible bearing upon tuberculosis of the lungs. Where there is considerable retraction of the left lung, for example, whether due to fibroid changes or to cavity formation, inspection frequently reveals an abnormal degree of cardiac pulsation. In very thin persons it may be possible to observe almost the complete cardiac contraction by means of the pulsations conveyed to the chest walls, as a result of the retraction of that portion of the lung which normally rests between the heart and the anterior wall of the thorax.

Disease of the lung is believed by some observers to be accompanied by changes in the overlying muscles, active disease causing spasmodic contraction, and healed lesions giving rise to loss of elasticity and wasting. It would seem advisable for the present to rely upon such changes in the symmetrical structure or mobility of the chest, rather than upon these somewhat uncertain trophic muscular phenomena.

*Mensuration* of the chest is used chiefly to detect the asymmetry of the two sides, a defect clearly demonstrable in many instances by charting the outline of the thorax by means of the cyrtometer or measuring it with a pelvimeter. This method has the advantage of recording the variations in the chest wall so that they may be kept for future reference. Various elaborate instruments have been devised for accurately recording the outline of the thoracic wall, but the use of the pelvimeter and the lead strip is much less expensive and simpler, giving records which are sufficiently accurate for practical purposes, if care be used in employing them.

The tracing for recording the general contour of the chest should be made preferably at the level which will just avoid the scapulæ, although it may be made at any level for determining asymmetry. The sternal attachment of the fourth costal cartilage usually gives a level affording a definite outline of the chest wall not distorted by the scapulæ. The center of the sternum is marked by a small cross at this level, and a corresponding point on the spine (usually the eighth thoracic spine) is determined by means of the pelvimeter, and this point also marked by a cross. The distance between these two points is determined by reading the scale on the pelvimeter, some of which instruments may be locked at the

desired point by means of a set-screw. These two points are marked on a sheet of paper, and a line drawn between them. One end of the lead strip is then placed at the posterior mark and moulded as closely to the chest as possible, working it against the chest from the back toward the front. When in close apposition the tape is marked where it crosses the ante-

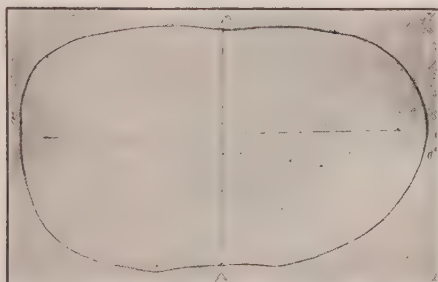


Fig. 10.

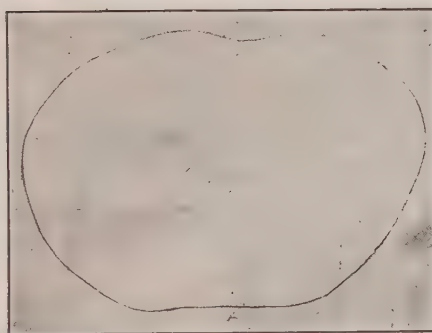


Fig. 11.

Figs. 10 and 11.—Chest tracings of two patients with left-sided pulmonary tuberculosis.

rior point, and carefully lifted away from the chest and laid on the paper with the anterior and posterior points of the tape corresponding to the points previously marked on the paper. A tracing is then made with a soft pencil, care being taken not to distort the lead strip, which should be held firmly against the paper.

It will be found that the point of greatest transverse diameter of the chest as measured by the calipers varies from

one-half to one and one-half inches (1.27 to 3.81 cm.) shorter than the tracing recorded by the lead strip. This is due to the fact that the points of the calipers can be pressed more closely to the ribs than with the cyrtometer, especially when there is considerable subcutaneous fat. It is well to record



Fig. 12.—Showing method of applying lead tape cyrtometer to obtain chest tracing.

on the tracing the actual transverse diameter as measured by the calipers, indicating it by a line at right angles to the antero-posterior line at the level of the greatest transverse diameter, shown on the tracing.

*Percussion.* There is considerable difference of opinion as to whether *percussion* or auscultation reveals the earliest evidence of tuberculous infiltration of the lungs. Early consolidation of

the lungs is manifested by various signs in different cases, depending upon the character and location of the tuberculous process. For this reason it is impossible to state which method of examination is the more valuable, as either method may first reveal the disease under varying circumstances.

To be of any value, percussion must be performed correctly, the blows of the percussing finger being made upon the

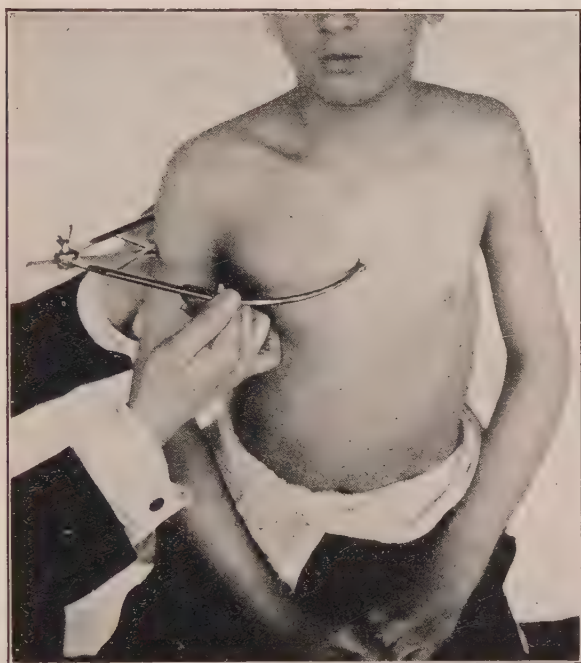


Fig. 13.—Showing method of employing pelvimeter for obtaining antero-posterior diameter of chest in taking tracing of the chest outline.

finger closely applied to the chest wall, only such force being used as will elicit an audible sound. Heavy percussion is not only valueless but misleading, and the method of using two or three fingers is to be especially condemned. The pleximeter finger should be rested evenly in the interspace to be percussed, but strong pressure should be avoided. The middle finger should be used for the plexor, the two distal phalanges being bent at right angles, and the blows being made from



the wrist or by the finger alone, the movement being made at the metacarpophalangeal joint. The plexor should not be allowed to press against the pleximeter finger after striking the blow, but should be quickly removed, and too many blows at one point should be avoided. One or two sharp, quick blows should be sufficient to bring out the sound. Slight impairment of resonance at the apices can be best elicited by percussing the chest from below upward, each interspace being compared with the corresponding point on the opposite side. A study of the apices by means of Krönig's resonant areas is frequently of value, slight infiltration at one apex being revealed by contraction of the isthmus on the side affected, or by what is equally significant, an obscuring of the dividing line between resonance and dullness, which is sharply defined in the healthy apex. To demarcate these zones of resonance at the apex very light percussion is necessary, the pleximeter finger being applied to the apex in a position at right angles to the clavicle. (Fig. 14.)

In percussing the chest it is important that the patient sit or stand in an easy position, the head held in the natural antero-posterior position, with the muscles of the neck and chest as relaxed as possible. In percussing the apices it is frequently found to be easier if the examining physician stand at the side of the patient or even posteriorly, in order to get the pleximeter finger in close apposition to the skin in the supraclavicular fossæ. For examining the back of the chest the patient should sit with the arms folded, the body bending slightly forward, with the shoulders relaxed and drooped. It is impossible to examine the upper portions of the lung posteriorly with the shoulders held rigidly erect. For examining the posterior portions of the lung the scapulæ should be pulled as far forward as possible in order to expose the posterior chest wall. To secure this it may be necessary to have the arms crossed in front with the hands resting well over the opposite shoulder. This position should never be assumed for the examination of the apices.

The most important evidence of early infiltration elicited by percussion is impairment of resonance, but a diagnosis of tuberculous disease should never be based on this finding alone. It must be considered only as a part of the general

clinical picture. Hyper-resonance or tympany (Skodaic) occasionally is present where the disease is incipient, slight, and composed of scattered tubercles. When there is a distinct difference between the percussion sounds at the two apices, it is necessary in certain cases to differentiate between an impairment of resonance at one apex or a hyper-resonance at the other. While this may be difficult, it usually may be deter-



Fig. 14.—Krönig's isthmus in a patient with an old tuberculous process at the top of the left lung.

mined by comparing the sound obtained at the apex with that elicited over the lower portions of the chest.

There are numerous sources of error in interpreting signs at the apices by percussion. These may be due to faulty technic; distortion of the chest by spinal curvature; inequality of muscular development; lack of knowledge of the normal size and location of the lung; or the normal difference between the sounds at the two apices. In addition to these potential sources of error, it must be remembered that impairment of

resonance results from a decrease in the amount of air-bearing tissue, and that this may be due to contraction of the apex as the result of scarring from a healed process, or probably from collapse induration as a result of dust inhalation in mouth-breathers. In persons who lead sedentary lives the percussion sound at the apices is practically never so resonant as in those whose daily life calls for exercises demanding deep breathing.

In the later stages of the disease percussion is of the greatest possible value in determining the extent and density of the infiltration, the presence of cavities, and the development of pleural complications, such as effusion and pneumothorax.

*Auscultation*, when properly performed, is a method of examination invaluable in detecting early infiltration, and in determining the character of the pulmonary process.

Before listening to the chest the patient should be instructed to breathe slowly, and slightly deeper than normal, with the mouth open. The respiratory movements should be easy, without undue muscular effort, and the movements should not be suspended at the end of inspiration or expiration, but an effort should be made to follow the natural rhythm. The examination should always be made in a room comfortably warm, for the least shivering of the patient may give rise to confusing sounds. The type of the binaural stethoscope to employ depends upon the personal preference, but in the majority of instances the instruments with a small hollow bell, without a diaphragm, will be found most satisfactory. Care must be used to see that the opening of the bell is held evenly and firmly against the chest, inasmuch as the slightest movement of the instrument on the chest wall may provoke very misleading sounds.

For a proper appreciation of the various changes in the breath-sounds, it is absolutely essential that the observer be thoroughly familiar with the normal respiratory murmur, and that he listens to the chest at every opportunity. It is impossible for the average physician to detect slight changes unless he is accustomed to listening to the breath-sounds almost daily.

The changes which occur in the breath-sounds in early tuberculous disease of the lungs are not pathognomonic, and must be considered only in their relation to the apex and in

relation with the other signs and symptoms. Healed lesions at the apex may give signs indicative of actual tuberculous disease, the true condition only being determined in some cases by other measures for estimating the presence of active disease, such as a study of the temperature and pulse.

The types of breathing suggestive of the presence of pulmonary tuberculosis are: feeble breathing, granular breathing, cog-wheel breathing, bronchovesicular, and bronchial breathing.

In the earliest cases met with clinically the first two mentioned are the types most frequently encountered. When the two last-named types are present, the condition can hardly be considered as an early lesion. Feeble breathing is only of value when localized over one apex and uninfluenced by deep respiration or by coughing. At times the breath-sounds may be absent. The breath-sounds are of a character which might be more correctly termed "indefinite" rather than by the name "feeble." True feeble breathing must not be confused with the areas of feeble or absent breath-sounds occasionally audible in patients in which the bronchi contain large quantities of secretion, and in whom the breath-sounds are again heard after coughing. Granular breathing is characterized by a fine, dry, sputtering quality of the breath-sounds which has been described as resembling the sounds of frying fat or as though soft granules of fine wet sago were being rolled over each other. It always suggests the addition of fine dry râles to the breath-sounds. While some observers believe that this type of breathing occurs only in the presence of early, active disease, others believe that it may occur in healed tuberculosis. While admitting the possibility of granular breathing in the presence of a healed lesion, its occurrence at one apex usually indicates active, recent pulmonary tuberculosis.

Formerly considerable weight was attached to the occurrence of cog-wheel breathing. When confined to one apex it may indicate the presence of a lesion, but is an uncommon finding in early tuberculosis of the lungs. This type is commonly met with in nervous individuals, especially women, in which case it is heard over the greater portion of the chest. Cog-wheel breathing may also be heard during an attack of acute pleurisy.



Prolongation of the expiration alone rarely may occur over an area of infiltration, but when present it is usually elevated in pitch, and has more or less the character of the broncho-vesicular type of breathing.

Pure bronchial or tubular breathing is heard in its most typical form in absolute, superficial consolidation of the lung. This is rarely met with in pulmonary tuberculosis, except in acute tuberculous pneumonia or in the scattered areas of the rapidly spreading broncho-pneumonic type. It is more commonly found in association with vesicular breathing, constituting broncho-vesicular breathing, the extent to which the bronchial element predominates being dependent upon the density of the underlying infiltration and its proximity to the surface.

Information of considerable value in determining the presence of abnormalities may be obtained, according to some observers, by comparing bilaterally the inspiratory or expiratory phases of the respiratory murmur. In this method of study the stethoscope is applied to the chest only during inspiration, for example, removed from the chest at the end of inspiration, and then applied to the corresponding point on the opposite side during the corresponding phase of respiration. By this means it is believed that slight differences on the two sides are more easily recognized than when one compares the entire respiratory murmur. It appears to the writer that the moving from one side to the other would tend to distract the examiner, and to prevent that concentration upon the breath-sounds which is so necessary if one is to obtain all the information possible.

When listening to the breath-sounds in the chest, all distracting noises and movements should be eliminated, the examiner training himself to concentrate the attention upon the breath-sounds alone, disregarding all adventitious sounds arising within the chest.

*Râles* are extremely valuable aids in the diagnosis of tuberculosis when their significance is fully appreciated. They are one of the signs which are liable to vary from day to day, and for this reason are of value in estimating the progress of a case under observation. They are very variable, however, and too much reliance must not be placed upon their increase or de-



crease, unless repeated examinations have demonstrated that the change is permanent.

Many cases are encountered in which no râles are evident on examination by the usual methods, the adventitious sounds becoming apparent only after coughing. It is always a good rule, in examining for pulmonary tuberculosis, after completing the regular examination, to go over the apices carefully again, after having the patient give a slight cough immediately followed by a deep inspiration. By this method small areas of fine râles may be discovered which would otherwise escape notice, especially in the "danger zone" along the internal border of the scapulæ at about the level of the spine of the scapula. Properly to expose this area, it is necessary to throw the scapula as far outward and forward as possible, by placing the arm far across the anterior chest, the hand resting well over the opposite shoulder.

Generalized râles have very little significance from the standpoint of tuberculosis, for it is only when they are localized to one or both apices that they are of value.

The information to be obtained from a study of the râles, in addition to the knowledge derived from the fact that they are localized to the apex or most marked in that region, consists in the size of the bronchi involved as indicated by size of the râles, and in some instances the degree of infiltration of the intervening lung tissue may be estimated from their character. That the finest bronchioles or alveoli themselves are implicated is usually indicated when crepitant râles are present, moisture in the bronchioles giving fine, moist râles, and in the larger bronchi râles of a larger size. The sibilant and sonorous râles do not have the same significance as the moist râles, as they are due either to simple swelling of the mucosa or to constriction of the bronchial lumen. Cavities are usually accompanied by bubbling râles—suggesting a space larger than any bronchus normally present at the point of examination. The point must always be borne in mind that râles of a certain size might indicate the presence of a cavity if heard over one portion of the lung, and not when heard over another portion, depending upon the size of the bronchi which should be present normally in that location. When a cavity exists the râles may indicate that fact by their amphoric quality.

Râles occurring in bronchi surrounded by a zone of consolidation or dense infiltration are, as a rule, most metallic and resonant, being loud and distinctly transmitted to the chest wall. The quality of the râles is especially important in determining the extent of the infiltration in those cases in which the râles are so numerous as to obscure the bréath-sounds.

It will be seen that the présence of râles may convey a great deal of valuable information, not only in the early stages of the disease, but also in determining the location and character of the infiltration, and as an indication of the progress of a case under treatment.

Vocal resonance is not of a great deal of value in early pulmonary tuberculosis, and only in rare instances will it supply information which cannot be more accurately obtained by other methods of examination. The transmission of the whispered voice is much more valuable, especially for localizing areas of consolidation or cavities. The whispering pectoriloquy heard over cavities usually possesses a more amphoric or cavernous quality than that due to a consolidation, which is usually more distinct and tubular. When the consolidation is near the surface, the whispered voice is beautifully clear and sharp, the words being distinctly transmitted to the examining ear, and one is impressed with not merely the sense of articulate speech, such as commonly accompanies deeper consolidations or cavities.

The early diagnosis of tuberculosis by means of the Röntgen ray has proved very disappointing, as it was hoped that by this means it would be possible to detect lesions in the lungs in the earliest stages. The more advanced the lesion, the more distinctly is it recorded upon the plates, but it is impossible to ascertain by this method an accurate idea of the activity of the process. The main value of skiagraphy is to confirm the results of physical examination in localizing the tuberculous process in the lungs, and to add certain information as to conditions in the chest which may be discovered only with the greatest difficulty by the ordinary methods.

In order to obtain a correct idea of the location and character of any intrapulmonary condition, it is practically essential that stereoscopic plates of the chest be made, the ordinary single-plate method of study being extremely unsatisfactory.

No greater mistake can be made than to turn a patient, in whom one suspects pulmonary tuberculosis, over to the röntgenologist for a diagnosis. Every physician who is doing much chest-work should take every opportunity to study personally  $x$ -ray plates of the chest, so that he may become familiar with the normal picture, and be in a position where he can appreciate the significance of slight abnormalities. This is not always possible, and in many instances it may be necessary to have the röntgenologist interpret the plates. Under these circumstances one must not accept too literally or too implicitly the diagnosis based upon the skiagrams. At its best the  $x$ -ray reveals only infiltration of a certain degree of density, and the more connective tissue or calcareous deposits there are the more intense the shadow. One may find oneself in the position of giving a bad prognosis in certain cases on account of the extent of the lesions, as revealed by this method, in a case in which the disease is practically arrested.

A good, safe rule, when one desires to have a patient studied by the  $x$ -rays, is to select an experienced, conservative röntgenologist, and have him make stereoscopic plates and study the case with the fluoroscope. Either examine the plates oneself, or have him make a complete report on the location of the process. Having learned the evidence desired from this method of study, it should only be considered as one sign or symptom, and interpreted only in its relation to the other available details of the clinical picture.

For determining the presence of small collections of fluid in the pleura, deep-seated abscesses, or collections of pus, localized pneumothorax and similar processes, the  $x$ -ray has considerable practical value, and in many obscure conditions the aid which it supplies may be unquestionable; but it must be confessed that in the majority of cases of pulmonary tuberculosis in which one desires assistance from this method of study, it will prove valueless or misleading.

#### TREATMENT.

The plan of treatment to be followed in the individual case of pulmonary tuberculosis varies greatly with the character of the infection. Whether this is determined by the virulence

of the infecting micro-organism, the resistance of the infected individual, or a combination of both, does not materially affect the question. That the disease gives rise to different pathologic conditions and clinical features in different cases is a fact of the greatest significance, and it is necessary to be able to distinguish between the various clinical types in order that one may select the appropriate line of treatment for the form of the disease in question.

From the standpoint of treatment, no diagnosis of pulmonary tuberculosis can be considered complete which does not include the extent of the pulmonary lesion, its physical character, and its degree of activity or quiescence. It is the failure to recognize the different requirements of the patient with a quiescent fibroid process confined to one apex, for example, and those of the patient with an active lesion of one or more lobes, with dense infiltration, and possibly breaking down of the tuberculous process, that is largely responsible for the unfavorable results obtained in the treatment of the disease.

The character of the disease varies between the rapidly extending process with a tendency toward caseation, most commonly seen in young individuals, and the fibroid process of a slowly advancing nature and no tendency toward consolidation, which ordinarily is accompanied by very slight constitutional symptoms, usually seen in older people. Between these two types examples are encountered which tend toward one or the other extreme, and these constitute by far the greater proportion of the cases met with clinically. The terms customarily applied to these extreme forms are, phthisis florida and fibroid phthisis, chronic ulcerative tuberculosis being applied to the intermediary types.

In addition to these general forms, there are exceptional types, such as tuberculous pneumonia and acute general miliary tuberculosis, the names of which are sufficiently descriptive. In phthisis florida the disease extends rapidly, without the least tendency toward walling-off of the process, and this results in the formation of large caseous areas and pneumonic patches, the former usually showing small areas of liquefaction and cavity formation in the older portions. The evidence of systemic toxemia in these cases may be ex-



treme: In fibroid phthisis on the other hand the process resembles fibroid disease of the lungs, with the changes associated with such conditions, the symptoms depending upon the location and extent of the process, and usually showing no toxemia, or but very slight evidence of the systemic inroads of the poison.

In the chronic ulcerative forms, as usually encountered, the disease may take on the characters of one or the other of these extreme types, depending upon the extent to which the tissues react to the tuberculous infiltration by the formation of fibrous tissue.

Tuberculosis of the lungs is manifested by two distinct processes, one consisting of the effect upon the lung by the tubercle bacilli themselves, and the other resulting from the toxins liberated by the bacteria at the site of the lesion, which ultimately gain entrance to the circulation. In certain individuals the pulmonary disease may be distinctly progressive, with very little evidence of systemic toxemia, and in others the pulmonary lesion may be very slight, and apparently fibroid, with marked evidence of disturbance of the general metabolism. In the following pages the word "activity" refers to the evidence of any effect of the tubercle bacilli upon the patient, whether localized to the lung or general in character.

For the proper treatment of pulmonary tuberculosis it is necessary that, in addition to determining the presence of the disease, the examination should establish the location, extent and character of the tuberculous lesions, and an accurate estimation of their activity. It is obviously important that the patient be carefully examined for the presence of any complications, whether of a tuberculous or non-tuberculous nature. In determining the degree of activity, it is necessary not only to make a thorough physical examination of the chest, but also to make a careful study of the patient's general condition, as shown by the weight, pulse, and temperature, and a careful consideration of the symptoms, such as cough, expectoration, gastric disturbances, pain, and general strength and well-being. In the treatment of the disease, as an index of the improvement or lack of improvement, the study of the patient's general condition gives much more accurate information than the physical examination, in the majority of cases.



**Prevention.** It is only right and proper that some reference should be made to the measures of preventing the disease before describing the methods of treatment, as in our present state of knowledge the various plans devised for preventing a tuberculous infection promise much greater returns than any known method of treatment of the disease after it has become implanted. In order to carry out intelligently the methods of prevention, it is essential for one to have a definite conception of the manner in which the disease is transmitted, the mode of infection, and the ultimate results of such infection in regard both to the invading micro-organism and to the infected individual. The entire problem of infection is far from settled, largely because of the impossibility of duplicating the natural conditions in experimental investigations. This is to be regretted, as whether infection takes place by ingestion or by inhalation, through the dried sputum or by "droplet infection," or by direct inoculation through the skin or mucous membrane, is not merely of academic interest, but is a vitally important question, which must be definitely answered before the prophylaxis of the disease can be carried out in an efficient and scientific manner. As to whether all primary infections take place in infancy or in early childhood, does not appear to the writer so important a question from the standpoint of prevention, unless it can be positively proved that secondary infection in adult life never, or hardly ever, takes place. In the light of our present knowledge, it seems absolutely necessary to make every effort to prevent tuberculosis by checking the spread of tubercle bacilli at the source, namely, the tuberculous individual or animal. This must be carefully carried out, either by insistence upon general preventive measures or by isolation of the infected individual. The most essential necessity in carrying out such a method of prevention is the early detection of the presence of the disease, and the weakest link in such a system is that the very nature of the disease makes its early detection difficult.

The sources of infection are tuberculous individuals, almost exclusively those suffering from the pulmonary form of the disease, and tuberculous cattle, although the latter have been shown to cause only a very small percentage of infections in human beings. Granting that only a relatively small propor-

tion of the infections are derived from bovine sources, the importance of preventing the sale of milk and meat from tuberculous cattle must be in no way minimized. The routine inspection and testing of cows used for supplying milk, and the rigid inspection of the slaughtering of cattle, provide a protection against the infection from these sources which cannot be disregarded.

The method by which bovine infection may be prevented is relatively well-defined, and presents no great difficulty other than that constituted by the magnitude of the problem and the necessity for organization, supported by legislation. The prevention of tuberculosis arising from human sources is extremely complex, and necessitates measures which involve nearly every branch of human interest or endeavor. This is particularly true, because our present methods of preventing infection at the source are lamentably inadequate, and because of this fact we must also direct our endeavors toward preventing the development of tuberculous disease in the infected individuals, or, if one does not accept the view of generalized childhood infection, toward building up the health and strength of the individuals, so that they may be able to resist infection when exposed.

The first and most important step in prevention lies in the early detection of the sources of the infecting micro-organisms, and the second consists in adopting such measures as will prevent the dissemination of the bacilli. This is the best that can be hoped for at present, as isolation of the tuberculous individual is, in the first place, too stupendous an undertaking, and, secondly, the insistence upon such a measure would defeat itself through causing the tuberculous to conceal the fact that they were suffering from the disease. For the carrying out of the various methods of prevention it is necessary to have the support of public opinion, and with this object in view a campaign of education is being everywhere carried out. In all educative campaigns great stress has been laid upon the widespread nature of the disease, its high mortality, and the infectiousness of the process. The public has therefore learned to view tuberculosis as a highly contagious disease, which has undoubtedly caused an enormous amount of hardships and suffering among its victims, and unfortunately also among

certain persons who are merely infected and not capable of transmitting the disease. It is to be regretted that the responsibility for this failure to make a distinction between the cases which are possible sources of contagion and those which are not rests to a great extent upon the medical profession itself, but this fault is somewhat excusable in view of the technical difficulties which frequently attend the differentiation of the two groups of cases.

Individuals suffering from tuberculous disease of the lungs may spread the tubercle bacilli by carelessness in expectorating the material coughed up, by the contamination of such articles as are placed in the mouth, or by small, invisible droplets expelled from the mouth during coughing, sneezing and laughing. Such people should be instructed in the measures necessary to prevent the dissemination of the infective material in the ways just indicated. The most important question is the proper disposal of the expectoration, the popular idea being to render it non-infectious by the use of disinfectants. The use of carbolic acid, bichlorid of mercury, and similar agents in receptacles in which the patient expectorates cannot be too strongly condemned. Their effect is merely upon the surface of the mucopurulent masses, leaving the central portion still infective, thus giving a false sense of security which may be responsible for harm. The best method is to have the patient use paper napkins, or small pieces of folded gauze, which can be used to cover the mouth during coughing, laughing, or sneezing, to expectorate into, and to wipe the mouth with afterward. The napkins should be used but once, and placed in paper bags, which can be handled safely, and burned at frequent intervals. Scrupulous personal cleanliness should be insisted upon at all times, frequent washing of the hands and lips, and rinsing of the mouth, being especially important. No male patient should be permitted to allow hair to grow on the face, on account of the opportunity it affords for collecting small particles of sputum. The patient should have his own dishes, table utensils, and drinking cups or glasses, which should be kept separate from those used by the other members of the household, and carefully boiled or washed separately each time they are used. It is also advisable that the patient have his own sleeping-

room, and certainly his own bed. While the patient is drowsy with sleep it is almost impossible for him to care for the sputum properly. For this reason it is advisable to have the bed-linen kept separate, and boiled before washing, as it may readily become soiled with sputum. This is especially true when the cases are advanced and confined to bed, in which case the bed-linen should be changed at frequent intervals. For the ambulant case nothing is so satisfactory as the paper napkin, which can be used and placed at once in a paper bag carried in the pocket for this purpose. Stiff pasteboard envelopes lined with some absorbent material also may be used to expectorate into, but their use usually excites an amount of attention to the act which the patients find objectionable. The expectorating into a paper napkin, which resembles an ordinary pocket-handkerchief, is much less conspicuous. The use of sputum cups, of either the pocket variety or the bedside forms with pasteboard inserts, should not be encouraged, as they are likely to become soiled, or the contents spilled, and, unless care is exercised, flies easily gain access to the sputum and distribute the infective material. The bedroom floor should have a wooden or oilcloth covering, to permit of frequent cleaning by scrubbing or damp-sweeping, never by dry-sweeping. When through accident any article becomes soiled by expectoration it should be cleaned with lye, and thoroughly scrubbed with soap and water, or if the nature of the material of which it is composed does not permit of this method of cleansing it should be sterilized by boiling. The room should be carefully screened, to exclude flies, and should be free of dampness, and so arranged as to permit a fairly general exposure to sunlight and fresh air (two most valuable means of destroying the life of the tubercle bacillus); there should be no dark corners where dirt might collect.

This brief outline of measures of prevention to be adopted for tuberculous patients does not, however, cover the entire subject, as one must also use every means possible for building up the health and strength of those in whom there is no tuberculous disease.

The public must, therefore, be instructed, not only in the means of prevention, but should be kept fully informed of the dangers of such predisposing factors as dissipation, child-



labor, unhygienic working and living conditions, bad housing, and insufficient nutriment. The education of the public along these lines is extremely important, as it is only by means of the pressure exerted by public opinion that legislation can be secured to control or correct the existing defective conditions.

It is impossible to estimate to what extent the spread of the disease is diminished by the early diagnosis and care of tuberculous disease, especially when the patients are segregated in a sanatorium or hospital during the period in which they are potential sources of infection. In planning a general campaign against the disease, this is one of the points which should not be overlooked; for while it is impossible to gauge accurately, or even approximately, how much is accomplished in the way of prevention by the early detection of the infectious cases, the elimination of a large number of infective sources must be of enormous value.

**General Considerations.** When the diagnosis of pulmonary tuberculosis has been made in the individual case, the question arises in the physician's mind as to which general method of treatment the case is best suited, whether the best environment would be at home, in a sanatorium, or in a hospital. The answer to this question depends upon the extent, character, and activity of the process, and upon certain economic and social factors. In a general way it might be stated that sanatorium treatment should be reserved for patients with slight active pulmonary involvement uncomplicated by tuberculosis in any other part of the body, and hospital treatment for those with extensive pulmonary tuberculosis, or slight involvement of the lungs, complicated by tuberculosis in some other part of the body, or by some other disease.

There are several exceptions to this very general rule, to be considered later, but both methods of treatment should be considered at best but temporary measures during the period of activity. The question of cure in the majority of cases is ultimately determined by the home surroundings, and by the conditions under which the patient works. The ability to stay in a sanatorium or hospital for a period of time sufficient to bring about a complete arrest or cure is only possessed by the exceptional patient. It is the failure to recognize this



fact which has given rise to the feeling that our present methods of treatment are entirely inadequate.

Sanatorium treatment possesses many advantages over home treatment, the most important being the following:

The patient finds it much easier to follow a method of living similar to that of those by whom he is surrounded. The living out of doors is much easier and more congenial, and the air is much less likely to be contaminated. A generous and well-regulated diet is more easily obtained than in the average home. The regulation of the amount and character of the rest and exercise is more easily controlled.

The mental stimulation derived from association with patients who have improved is of inestimable value. The patient receives an education in the methods of prevention and treatment, and obtains a general knowledge of the disease and its various manifestations and complications which it is almost impossible to secure in any other way.

The method has certain disadvantages, which must be taken into consideration, although they are more than counterbalanced by the advantages. The most serious disadvantages of a sanatorium régime are as follows:

The separation of the patient from his family, which, while usually a distinct advantage, may at times be a serious disadvantage, especially when he is so far removed as to make the visits of members of his family prohibitory. This is especially true of the more advanced cases. Occasionally the patient becomes mentally depressed from the association with so many sick people, this being mainly on account of the vicious habit, so common to sanatoria, and so difficult to suppress, of the patients discussing their symptoms among themselves.

The lack of individual attention is sometimes experienced by patients in sanatoria where large numbers of cases are treated, and where necessarily a certain amount of routine must be maintained.

The worst defect of the sanatorium lies in the fact that so much is done for the individual that there is a risk that he may lose that self-reliance so essential for his future welfare.

There are other factors which may influence the selection of sanatorium treatment, such as bad housing or living con-

ditions, lack of necessary care and attention at home, vicious associations or habits, etc.

Sanatorium and hospital treatment have the advantage of removing from the family a possible source of contagion during the period of infectivity. This is a point to be constantly borne in mind in regard to patients in whom for any reason there is cause to doubt their complete co-operation in the carrying out of the necessary preventive measures.

Hospital treatment may also be found temporarily necessary in patients in whom the disease has pursued an inactive course, during periods of acute exacerbation.

The home treatment of the disease is, after all, the most important for the average physician thoroughly to understand and appreciate, and for this reason the various measures of treatment will be given as they can be applied in the patient's home, where, with considerable patience and attention to detail, many of the advantages of the sanatorium can be secured.

There is probably no other disease in the treatment of which it is so necessary to exercise patience, perseverance, encouragement, and constant attention to every apparently trivial detail. It is frequently the little detail of treatment repeated over long periods of time which may finally determine the question of success or failure in the attempt to overcome this insidious and stubborn disease.

**Climate.** The value of certain climates in the treatment of tuberculosis is so firmly implanted in the minds of many medical men, as well as members of the laity, and the virtues of these climates are so frequently extolled by intelligent and reliable members of the profession, that it is with considerable hesitancy that one gives expression to views upon this subject which may not be in accord with those commonly held. When one comes to a careful consideration of the question of climate, one finds that there is a curious lack of uniformity in the various climates recommended in the treatment of tuberculosis, and that they do not seem to possess any physical property in common. One is forced to conclude that the claims of these various climates must rest largely upon the fact that the medical men in the various resorts, through their wide experience, and possibly by virtue of their personal in-

terest in the disease, have been better able to treat the patients, and probably have had them under better control than could be secured in their own homes. Equally good results seem to be obtained in sanatoria situated in regions for which no great claims are made, so far as climate is concerned, and where the only climatic factors they possess to recommend them is air uncontaminated by the dust and smoke of a large city. It must be recognized that the patients who have been under treatment for some time not infrequently show a temporary improvement under the stimulation of change in scene and surroundings. There are also certain parts of the country where, on account of climatic conditions, it is much easier for the patient in search of out-door life to pursue the mode of life so desirable, if not essential, for their recovery.

In deciding where to send a patient suffering from pulmonary tuberculosis, the most important considerations should be the skill and experience of the medical attendants, and the equipment for carrying out the necessary treatment, rather than any reputed climatic advantages of the location.

**Fresh Air.** It is not necessary to analyze or to attempt to determine the essential feature of fresh air, which makes it so valuable in the treatment of pulmonary tuberculosis. The fact remains that there is probably no one factor which is so important in the treatment of this disease as an unlimited supply of fresh air. It is very fortunate that so important an aid to treatment is so very easy to secure in practically an unlimited supply, although the very ease with which it is obtained probably is largely responsible for the extent to which it has been neglected in the past.

There are several points which must be emphasized in regard to fresh air, which may appear extremely trivial and yet are so important that they will bear repeating. The main point to be recalled is that the desired object is to obtain a supply of air to the respiratory tract in the greatest possible degree of purity and freshness, and at the same time to keep the patient comfortable. The first object is easily obtained by keeping the patient outdoors as much as possible, and seeing to it that when not outdoors there is a sufficient supply of fresh air indoors. In the localities where the houses are not too close together, sleeping porches may be utilized, or in lieu of this

an ordinary porch may be used, or even a yard or roof may serve the purpose. A room well supplied with windows, kept open as far as possible from the top and bottom, or with the sashes removed, answers almost as well as the sleeping porch. Some patients object to the draught caused by open windows, under which circumstances considerable comfort may be derived from the use of a small screen which will prevent the air from blowing directly upon the patient, the comfort of whom must be respected, if one expects him to persevere in living in the fresh air. One must, therefore, see that the bedclothes are sufficiently heavy, and that the bed is made properly. In very cold weather it is advisable to see that there is adequate protection beneath the mattress as well as over it, and, where the bed is exposed, a light rubber covering should be provided for stormy weather. The so-called "Klondike bed" is a very convenient and comfortable method of making the bed during the extreme weather, as it not only provides blankets beneath as well as over the patient, but the blankets are arranged so as to prevent any air leaking in around the edges. Sleeping-bags made of special blanket material are preferred by many patients, as they are less likely to become disarranged by moving about in the bed. The object to be obtained by such sleeping accommodations is the exclusion of the cold air, and to keep the bedclothes as close to the patient as possible, so that it will not be possible to dissipate much heat by warming a large air-space beneath the covers. In cold and damp weather the bed should be dried and warmed before the patient gets into it, otherwise there may be suffering from the chilling. The patient must wear sufficient underclothing at night, and a woolen nightcap or similar covering for the head is almost essential when sleeping out of doors. When sleeping outdoors such patients as require absolute darkness in order to sleep may resort to an opaque bandage applied over the eyes. Where the sleeping-quarters are limited, and the necessity arises of keeping the remainder of the room warm, a window tent will be found to be a great convenience, as the head of the bed may be placed outside the window, or immediately beside it, and the fresh air supplied directly to the patient's face and excluded from the remainder of the room. These window tents may be purchased, or made

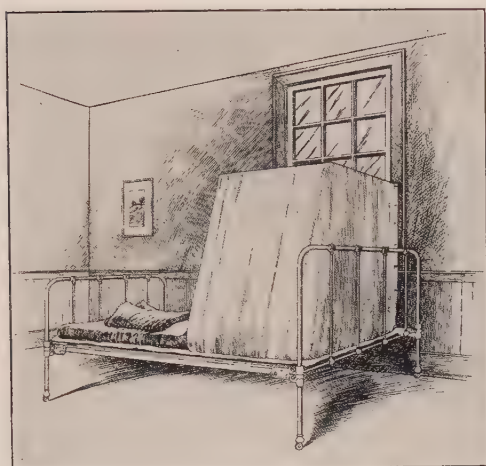


Fig. 15.

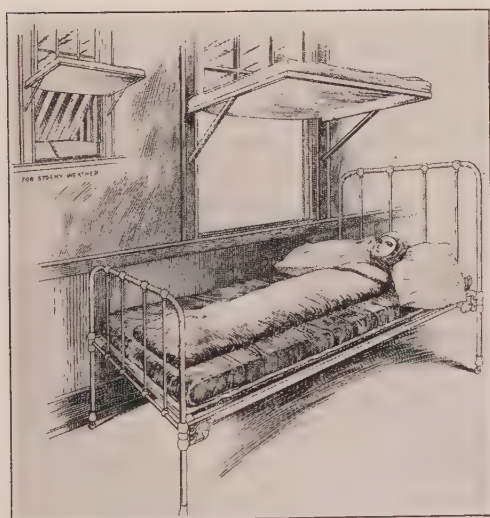


Fig. 16.

Figs. 15 and 16.—Showing home-made window-tent, open and closed. (Henry Phipps Institute, University of Pennsylvania.)



at home, as the patient's circumstances dictate. An inexpensive home-made window tent, which can be easily made out of heavy canvas duck, with a simple wooden frame, is illustrated on page 411. An awning on the outside of the windows offers protection from wind, sun, rain, or snow, and insures privacy.

For resting outdoors, "taking the cure," as it is called, an easy reclining chair is necessary, a steamer chair or Adirondack reclining chair answering the purpose very well, with the patient well protected against the cold by means of steamer blankets or sitting-out bag. The patient's feet must be warmly and loosely covered when sitting out, the ordinary tight leather shoe not being suited to this purpose. A warm room in which to dress and undress, bathe, and eat is an additional indispensable comfort to the patient living outdoors.

The patient with pulmonary tuberculosis should be kept in the open air or fresh air the entire twenty-four hours, except for the time necessary in which to dress, undress, eat, and bathe, with an occasional half-hour for writing, sewing, or duties of like nature which cannot be performed in the open air in very cold weather.

Most of the suggestions for the comfort of the patient refer to very cold weather, and yet it is just as important to look after the patient during the warm days. The patient must be protected from the direct rays of the sun at all times when sitting out, except when otherwise ordered by the physician, must not be wrapped up too much for comfort, and is to be protected from mosquitoes, flies, and other warm weather pests.

**Rest and Exercise.** In the management of a case of tuberculosis, rest and exercise are two extremely valuable aids, when properly regulated in the individual case. While rest may be rarely applied improperly, so that a patient may possibly derive harm from its abuse, it is exercise which is usually misapplied, and too mistakes on this score are referable great possibilities for injury to the patient. The importance of rest in any patient with active pulmonary tuberculosis is beyond question, and by "rest" is meant absolute rest in bed during the entire twenty-four hours. It is even advisable to put to bed every patient with pulmonary tuberculosis when

first coming under treatment, until they have been kept under observation for a sufficient length of time to determine their temperature and pulse when at absolute rest. One is then in a very much better position to direct the future treatment of that individual patient, and it also provides an index with which to compare the effect of treatment.

When to discontinue the absolute rest in bed, and to allow the patient to get up, dress, and sit outdoors, is a question which must be determined in each individual case. In the face of a subsidence of the original physical signs, a gain in weight, a pulse-rate approximating 100 in the afternoon, with fever not above  $99\frac{1}{5}^{\circ}$  or  $99\frac{1}{5}^{\circ}$  F. ( $37.5^{\circ}$  or  $37.7^{\circ}$  C.), and an absence of all complications, the patient may be allowed to sit outdoors, or to "take the cure," as it is called (Fig. 17, B).

The necessity of an accurate daily record of pulse and temperature in the early part of the treatment cannot be too strongly urged, since these data supply information absolutely necessary for the intelligent treatment of these cases. A few precautions may not be amiss in reference to the taking of the pulse and temperature. The pulse should be taken after the patient has been resting for at least one-half hour, and this also applies to the taking of temperature. The temperature should never be taken directly after the patient has been drinking anything hot or cold. The thermometer should be placed well under the tongue and retained in the mouth, with the lips tightly closed, for ten full minutes. It does not make any difference whether the thermometer is a "half-minute" or a "two-minute" thermometer, it must be kept in the mouth ten minutes, as with so much depending upon such a slight variation as one degree above normal, or less, no thermometer can be depended upon to register accurately in less than ten full minutes.

The stage at which the tuberculous patient may be permitted to exercise, is an extremely important question to decide, and one for which it is difficult to lay down fixed general rules. It is really a question to be decided in the individual case, the decision being formed by the extent and character of the pulmonary lesion, the presence or absence of complications, the duration of the cure, the weight and general condition of the patient.

In a broad way, one might state that no patient should be allowed exercise so long as there is evidence of active lesions in the lung. Where no contraindications exist, the pulse-rate and the temperature are the best guides in permitting exercise, and controlling it after it has been allowed. A good conservative rule, to which there are certain exceptions, is not to allow any patient to take exercise until the afternoon temperature (or maximum daily temperature) is below  $99^{\circ}$  F. ( $37.3^{\circ}$  C.), and the pulse-rate 90 or less when at rest (Fig 17, D). The exercise should be discontinued if the temperature or pulse show any tendency to rise, when taken at the end of one-half hour's rest after the exercise. The exercise should also be discontinued upon the appearance of any unfavorable symptom or sign, such as loss of weight, dyspnea, hemoptysis, pleurisy, fatigue, etc. There is a fairly large group of patients who do not present a high afternoon temperature, and in fact the afternoon temperature may be normal, but in whom the morning temperature is markedly subnormal (Fig. 17, C). One must be extremely cautious in permitting exercise in these cases with a wide daily variation in temperature until the morning temperature more nearly approaches normal (Fig. 17, D). These cases usually have a high pulse-rate, which will serve as a warning, but is not always true, for the pulse at times is relatively slow.

Having determined the advisability of exercise, the question arises as to the form in which it is to be given and how far it is to be pushed. Undoubtedly the best form of exercise in the early part of the treatment is walking, and at first this should be allowed only for five or ten minutes a day, until it has been demonstrated that the exercise is not followed by any deleterious effect, as evidenced by the temperature and pulse. It has also been suggested that the blood-pressure may serve as a guide to the advisability of continuing the exercise, a slight drop in the blood-pressure following a rest-period after the exercise, or a decided drop in pressure immediately after the exercise, being an indication that the activity should be diminished. Even a slight drop is to be considered a sufficient contraindication to an increase of the amount of exercise. While it has been suggested that the blood-pressure serves as an excellent guide, the temperature and pulse-rate, if accu-

rately recorded, should provide one with sufficient information to gauge accurately the amount of exercise to be permitted. If the temperature and pulse, taken one-half hour after the walk, show no distinct increase, the walk may be lengthened from two to five minutes daily, until the patient is taking from one to three hours' walk. It may usually be found beneficial to supplement the walk with work of some form, whenever the walk has reached two hours, in order to provide some interest to the exercise, for walking soon may become very monotonous. The main objection to games in the open air, for arrested cases, consists in the fact that the interest excited by the game,

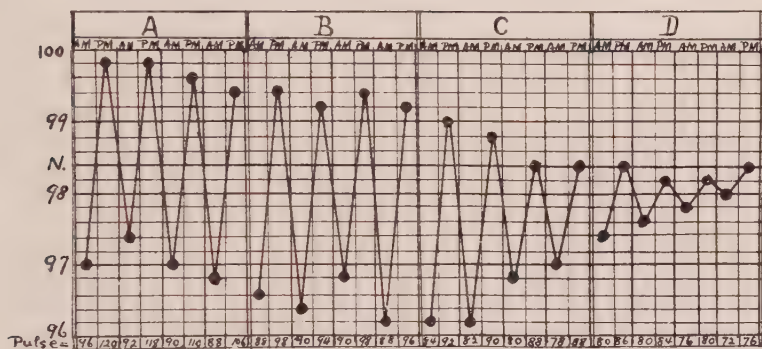


Fig. 17.—Showing various types of temperature records met with in different stages of tuberculous disease of the lungs (see text).

especially if there is any competition involved, leads the individual to overexert himself, or to become overfatigued. The problem of providing amusement for the patient on rest is a very serious one, as it is difficult for the patient to keep his mind occupied with such games as chess, checkers, parchesi, and cards. Sewing or crocheting may be permitted in moderation, and reading, if some supervision is maintained, so that reading books of too depressing or of too serious a nature is avoided. Studying should be absolutely prohibited until the patient is on a considerable amount of exercise. The only points to be borne in mind in selecting the work are, that the work should be as much as possible in the open air, should always be stopped short of fatigue, and should not



require any violent straining effort. There is no reason why an early case of pulmonary tuberculosis should not gradually work up to eight hours exercise a day, and it is beyond question that exercise, when properly controlled, has an extremely beneficial effect upon the patient's general condition. There is one point of extreme importance, and one which does not receive the attention it should, namely, the very injurious results which may follow deep-breathing exercises. So long as there is any activity, or any reason to suspect activity of the tuberculous process, all deep-breathing exercises should be absolutely forbidden. Deep-breathing as a health measure, has been so frequently recommended to the laity, especially where there is any reason to suspect pulmonary disease, that patients will frequently adopt this procedure without the advice of their physician. This fact should be constantly remembered, and patients warned of the dangers resulting from this apparently harmless form of exercise. While not going so far as to say that deep-breathing is harmful or useless in healthy individuals, or in people with no active pulmonary disease, one must admit that the value of the measure is certainly very much overrated. Anyone who has had under observation many cases of tuberculosis with extensive pulmonary invasion must have been impressed with the relatively small amount of functioning lung tissue necessary to meet the ordinary demands of life. The dyspnea occasionally encountered in this disease does not appear to be dependent upon the amount of lung tissue affected, but upon some other factor, many of the worst cases met with being patients with only a relatively small crippling of the aërating surface of the lungs.

**Diet.** Fresh air, rest, and exercise do not, as a rule, offer any very serious problem in the average cases, although they require careful control, and the exercise of judgment in their application. To secure a well-balanced, varied, nutritious, digestible diet in sufficient quantity, will frequently tax the patience, perseverance, ingenuity, and resourcefulness of the physician in attendance. In institutions for the treatment of tuberculosis it is advisable to study carefully the amount of food per patient consumed daily, and accurately to measure the food used, so as to determine the number of calories each patient is receiving daily, and the proportion of protein, fat,



and carbohydrate. This is necessary, not only that we may learn what is the diet which seems to give the best results in the majority of cases, but also to ascertain how that diet may be provided most economically. In practice, however, it seems best, in view of our present lack of knowledge, not to attempt the weighing of the patient's food, but to provide that diet which seems best suited to the needs of the individual. If one should be interested in determining, for comparison, the caloric value of the food consumed, and in estimating the proportion of protein, fat, and carbohydrates consumed daily, it may be stated that according to the most reliable studies we possess the total value for one day should be about 3200 calories, with about 100 to 130 grams (3 oz. 230 gr. to 4 oz. 255 gr.) of protein, 100 to 110 grams (3 oz. 230 gr. to 3 oz. 384 gr.) of fat, and carbohydrates sufficient to bring the diet up to the required value. It would be impossible to attempt to describe in detail the various articles of diet which might be resorted to in various conditions. The foods which have proved of most value in the majority of cases form the so-called milk-and-egg diet. This possesses so many advantages that it should be given a fair trial in every case, and not discarded until it has been clearly proved unsuited to the individual patient. In the first place it is usually readily obtained, and, as a rule, is not extremely expensive; furthermore, milk and eggs can be accurately and easily measured by the patient, which allows of a ready increase or decrease in the quantity, and the patient can usually take them even when there is not very much appetite. Milk has the advantage of being a well-balanced food which does not tax the digestive powers of the invalids, and while the bulk is a distinct disadvantage, it is usually not a serious one. It is especially valuable for the reason that it may be given at relatively frequent intervals. A very common mistake in giving this diet, and one that has been largely responsible for many of the failures to obtain good results from its use, is the failure to limit the amount of solid food consumed while the patient is taking large quantities of milk and eggs.

It is extremely important that the patient be instructed carefully about the amount of milk and eggs to be taken, and also be told just when to take them. It will be found most

satisfactory to write out for the patient a daily diet schedule, with the quantity and time for taking accurately recorded. In this way mistakes can be avoided which it is possible may prove serious in some cases. It is difficult to outline in detail a diet satisfactory for every case, but the effort will be made to give a daily diet schedule which will at least serve as a basis for treatment, with such modifications as may be needed in the individual case. It is customary in referring to the diet to speak of a "twelve-six" or an "eight-four" diet, or some such combination of figures, the first figure referring to the number of glasses of milk taken daily (reckoning four glasses to a quart), and the second to the number of eggs. In the average patient it will be found best to start in with eight glasses of milk, four eggs, and but one meal daily. If it is found that the patient gains weight upon this quantity of food, and hunger seems to be satisfied, it may be maintained, or the milk and eggs can readily be increased if there is a failure to gain in weight. The disadvantage of starting the patient with a "twelve-six" diet is that it may be found too much for proper assimilation, and may create a distaste for the diet which it may be impossible to overcome. A sample dietary of a patient on an "eight-four" diet would be as follows:

7.30 A.M. Two glasses of milk and two eggs.

10.00 A.M. Two glasses of milk.

1.00 P.M. Dinner—Meat and vegetables; fruit for dessert  
by preference.

4.00 P.M. Two glasses of milk.

6.30 P.M. Two glasses of milk and two eggs.

When the patients are not exercising they frequently do better if a longer interval is allowed to elapse after the mid-day meal, the two afternoon feedings being given at 5 and 7.30, or at 6 and 8.30 P.M. The milk may be given cold or at room temperature, as the patient prefers, even being slightly warmed if desired. The milk should be swallowed slowly, and not gulped down in large quantities, or merely sipped. In very cold weather it will usually be found that the patient will appreciate very much a warm drink in the morning, such as weak coffee or one of its substitutes, or hot cocoa.

The method of taking the eggs may usually be left to the preference of the patient, who usually finds it most satisfac-

tory to swallow the eggs whole, or to swallow the yolk and white separately, without breaking the former. This method has the advantage that the egg is practically tasteless, which will be found to be a great advantage.

The swallowing of the eggs may be facilitated by placing them in a glass and covering with a small quantity of milk, orange-juice, or something of the kind. If the patient prefers to take the egg in the milk, they should be well mixed by shaking together, and then strained before drinking. If possible they should be taken with very little, if any, flavoring, preferably not sweetened, and the common practice of adding sherry, whiskey, or brandy to the milk-and-egg mixture, should only be resorted to in exceptional cases.

Some patients have a firm belief that they are unable to take milk, believing that it does not agree with them. An effort should be made to alter this view if possible, assuring them that not infrequently people suffer from gastric and intestinal disturbances for the first week or two before the gastro-intestinal tract becomes accustomed to taking care of that form of food. If the prejudice is very strong, milk may be given in very small quantities, a few ounces, once or twice a day and gradually increased in quantity, as the patient finds he suffers no ill effect. The whole-milk should be used by preference, but occasionally it may be found necessary to resort to buttermilk (prepared from the whole-milk), koumiss, or some similar beverage, and, as a final resort, the malted milk preparations may be tried for a short period, as a substitute for the raw milk.

Occasionally patients will be encountered who are unable to take the eggs, even after repeated attempts. Sometimes they can take the eggs if cooked, but even then it will be found impossible to give them in the same quantity as when taken raw, and the patients soon tire of them. When the patients are unable to take the eggs, or it is found impossible to secure eggs that are absolutely fresh, certain changes have to be made in the dietary to provide sufficient nutriment. Solid food will have to be provided at the morning and evening meals, to take the place of the eggs, allowing a longer interval to elapse before taking food again than would be necessary after taking eggs. At times patients find it impossible to take the

milk-and-egg diet after persistent efforts, in which case it is advisable to resort to three meals a day, care being taken to see that an interval of four to five hours, between meals is provided. If it is found that the patient fails to gain on the ordinary food, the three meals may be supplemented by the addition of olive oil or cod-liver oil, and the patient advised as to the quantity and character of food to be taken, so that the meals may provide the greatest nutritive material for the amount of food taken. This is extremely important, as the average layman is woefully ignorant about the nutritive or caloric value of foodstuffs.

It might not be amiss to mention some of the precautions to be observed when three meals are taken, and although some of these may appear trivial and commonplace, their importance is such that they are worth repeating. Fried foods should be avoided, all foods being prepared by roasting, broiling, boiling, or baking. Hot cakes and hot breads of various kinds should never be taken, bread somewhat stale, or toasted, being by far the best way in which to take this form of food. Pastries also are very objectionable, the desserts to be preferred being custards, rice, sago, cornstarch, and similar preparations. Fruit also is a desirable article of diet at the completion of a meal. Some of the articles of diet which may be found to be of benefit are rolled oats, wheat preparations, cocoa, peas, beans, potatoes, rice, butter, macaroni, cornmeal mush, lamb, beef, chicken, fish, bacon, nuts, raisins, and dates. Soups should preferably be in the form of a purée.

While this list is extremely brief and does not in any way begin to designate every food which may be taken, the articles mentioned are the ones upon which special emphasis should be laid. The majority of patients must be instructed to eat slowly and to chew the food thoroughly before swallowing it. It is important to see that the teeth are in good condition, in order that the food may be properly masticated.

When the patient is taking three meals daily, and for any reason it seems advisable further to supplement the diet, if it is impossible for them to take milk, beef-juice may be given between meals, or even scraped beef. Most patients do not object to beef-juice if the meat is slightly broiled, quickly cut up, and placed in a press which has been heated, and the



receptacle also warmed, the juice being consumed before it has cooled. They seem to prefer it warm, and it is extremely difficult to warm the juice up afterward without precipitating the albumen, unless this is done upon a water bath. Some prefer the juice of the raw meat and the raw scraped beef, which may be given properly seasoned in the form of a cannibal sandwich, on thin stale or toasted bread. A point usually overlooked in the making up of a dietary is that as much variety as possible has a distinct advantage, (monotony being one of the serious objections to the milk-and-egg diet), as has also the flavor and careful preparation of the food. No detail is too insignificant in the ordering of the daily menu, and this subject should receive the closest attention of the attending physician. Frequently it will be found necessary to experiment with various varieties of foodstuffs before a dietary can be obtained which creates a gain of weight. Even when the full milk-and-egg diet is contraindicated, it will be frequently found that one or two glasses of milk can be taken with each meal, and this, of course, adds considerably to the daily amount of nutritive material consumed.

A point frequently overlooked in treating cases of pulmonary tuberculosis is that in the early cases only sufficient food should be given to assure a steady gain of weight. It is a mistake to try to force the feeding in those cases, in order merely to see how much can be gained in a short time. While a rapid gain in weight is very encouraging to the patient, it is frequently acquired at too high a cost, with a sacrifice of the future gastric power as a result. The natural method of eating for most people consists of taking three meals a day, as the stomach has been accustomed to receiving food of a certain kind at fixed intervals. No matter what method of dieting is pursued in the early part of the treatment, the main object to be kept in view is to get the patient back to three meals a day, and to have him hold or gain his weight upon such a diet.

The more advanced cases present a somewhat different problem, and it is in such that forced feeding, when given with judgment and careful supervision, is more permissible. The immediate need of increased nutrition in these cases warrants one in insisting upon their taking food in excess of their desire.



Children suffering from tuberculosis do not, as a rule, take kindly to the milk-and-egg diet, although it should always be given a careful trial before being abandoned. In cases of this age-period three meals a day will usually be found most satisfactory, especially when cod-liver oil is given in addition. They will frequently take the oil in the form of an emulsion without the slightest trouble, when it is impossible to make them take an increased quantity of food, and generally they do very well on this addition to their diet, and in consequence usually gain steadily in weight.

*Hydrotherapy.* In the treatment of a prolonged illness, such as is usually the rule in pulmonary tuberculosis, no means must be neglected to improve the general tone of the patient, especially such measures as are unaccompanied by any dangerous after-results or risks of harm. The application to the skin of water of varying temperature has long been one of the most popular and accessible measures for combating disease, and in certain conditions it has attained considerable and richly deserved reputation. In the treatment of all cases of tuberculosis the external application of water will prove of unquestionable value. It is not necessary to have the complicated apparatus of the hydropathic institution in order to secure favorable results. In early cases a cold chest-bath in the morning on arising usually gives the patient a sense of stimulation that cannot be obtained in any other way. In cold weather it is necessary for the patient to have a warm room in which to carry out this procedure, and it should always be followed by a brisk rub with a rough towel. Some cases find the chest-bath insufficient, in which case a general cool sponge or shower may be substituted, colder water being gradually applied until the desired effect is obtained. It is necessary to caution patients against taking baths with water that is too cold, and to warn them against unduly prolonging the application. The bath should be absolutely forbidden, if it is not followed by a definite reaction, with a general glow and sense of well-being. If the patient fails to react, and feels chilled for any period after the baths, they must be discontinued. Nearly everyone, however, can take the cold chest-bath with benefit, if care is exercised in the method of application.

The use of cool or cold sponges in the cases with excessive elevation of temperature is discussed elsewhere, also their employment in preventing night-sweats. (See pp. 456-7 and 458.)

For a patient with afternoon fever and a subnormal morning temperature, one must be especially careful in using cold baths in the morning. The majority of patients with this type of temperature obtain more comfort from a warm bath in the morning. The temperature of the water used for the morning bath may from day to day be gradually lowered until finally a cool bath may be employed, but, as previously stated, it should be discontinued on the slightest evidence of chilling.

For general purposes of cleanliness, the ambulant patient may be permitted to use the tub two or three times a week. The water should be comfortably warm, not excessively hot, and should always be followed by a cool or cold sponge. Only rarely should the bed patient be bathed in the tub; in most instances it is advisable to have it done in bed between blankets, and one extremity at a time being bathed, followed by bathing of the trunk, with not too much of the body being exposed at any one time.

The convalescent patient will occasionally ask for advice in regard to ocean or surf bathing in the summer time. While a quick bath in the salt water would probably prove of value, the discomforts usually attending this form of bathing, the temptation to remain in the water longer than advisable, and the fatigue from resisting the force of the waves, more than counterbalance any benefit that might be derived from it. When the patient has reached the point where it is possible for the physician to consider him an absolute cure, say, after the disease has been arrested for two or three years, this form of diversion may be practised, provided that every precaution is observed to prevent prolonged chilling and over-fatigue.

*Artificial Pneumothorax.* During recent years this method of treating pulmonary tuberculosis has been extensively applied, and the results carefully recorded and studied. Theoretically, this placing of the lung at absolute rest should be followed by arrest of the process in the large majority of cases, but unfortunately in practice this is not the case, the main reason being that only a small proportion of the cases in which it is tried are suitable for this method of treatment. The suc-

cess following the production of the artificial pneumothorax will be directly proportionate to the care and judgment exercised in the selection of cases in which it is induced, as the majority of observers believe that only about 5 per cent. of all cases are suitable for this form of treatment, and it is a procedure that is not without certain disadvantages and risks. The injections must be kept up for a long period of time, during which the patient should be kept under absolute control, preferably in a sanatorium, making it an expensive and tedious method of treatment. The dangers of air embolism, pleural shock, subcutaneous emphysema, and injury to the lung, while probably more theoretical than real, cannot be ignored, as the procedure possesses a slight mortality due to the injections. This is a truth to be taken into account when considering the advisability of instituting this method of treatment.

The development during the treatment of serous effusions, which may later become purulent, is of relatively frequent occurrence, and constitutes a very grave complication which may arise in the course of the treatment in any patient, and appears to bear no relation to the care exercised in making the injections. Taking everything into consideration, it seems that this procedure should be resorted to only in those patients who have failed to respond to general hygienic treatment, in whom the rapidity with which the disease spreads renders delay dangerous, or in whom there is some special indication such as hemoptysis. Only those cases are suitable for treatment which present certain generally accepted indications, a list of which is given by Sloan, as follows:

“(a) Gross and active lesions in one lung, with a quiescent lesion not extending below the level of the fourth rib in the other lung; (b) gross and active lesions in one lung, with a mildly active lesion not extending below the level of the third rib in the other; (c) quiescent lesions, bilateral, but suitably located, with a history of aggravating cough and profuse expectoration; (d) arrested but suitably located lesions, with a previous history of collapse whenever work was attempted; (e) arrested lesions, but with a history of recurring hemorrhages.” He considers as unsuitable all cases showing: “(a) Gross and active lesions extending below the level of the third rib on both sides; (b) an extensive gross lesion in one lung,

and a lesion located at the base in the other; (*c*) serious complications, such as cardiac disease, arteriosclerosis, ulcerative laryngitis, chronic diarrhea, extensive tuberculous ostitis, and nephritis, acute or chronic; (*d*) disease apparently of long standing, as shown by marked fibrosis of the lungs, thoracic deformities, decided cardiac displacement and dyspnea; (*e*) a history of chronic alcoholism; (*f*) a history of recurring hemorrhages from both lungs; (*g*) a marked emphysema; (*h*) an erratic or excitable temperament; (*i*) real or apparent old age."

It will be noted that he advocates employing this method in cases in which the disease is bilateral; and while he is supported in this opinion by many careful observers, the writer cannot help but feel that from a theoretical standpoint bilateral disease should be considered as a contraindication, a view which a limited experience in the employment of this procedure has tended to confirm. If ever employed in a case of bilateral disease, the uncompressed side certainly should be most carefully watched for any evidence of increased activity. The ideal case for treatment by this method would be a patient with an acute unilateral tuberculosis, tending to extend or to show no signs of becoming arrested under careful general treatment; in addition, there should be no complications, and the disease must be so limited as to warrant the persistence of a fair area of air-bearing lung tissue. It must be confessed that such cases are not commonly encountered. At times it may be justifiable to employ this method of therapy in certain advanced cases, with the object of ameliorating the symptoms and securing additional comfort for the patient, without any thought of arresting the disease.

The immediate results of this treatment are usually very good, and at times almost miraculous, transforming within a few days a patient with a high fever, profuse expectoration, poor appetite, and digestive disturbances, in whom the disease is steadily progressing, into one who has no fever, or very slight pyrexia, very little expectoration, a good appetite, no gastric disturbances, and a general sense of well-being and comfort. While usually the injections are very well borne, occasionally the injection of the nitrogen gas into the pleural space is followed by dyspnea, considerable general distress, and severe pains lasting several hours.



The ultimate results are not so encouraging, even in selected cases, for the majority of writers report ultimate recoveries in only about 50 per cent. of the cases. It will thus be seen that, while of considerable value for ameliorating the symptoms in certain advanced cases, as a method for curing the disease it is a procedure whose field of application is distinctly limited.

*Technic.* It is impossible to describe in detail the various methods employed for inducing artificial pneumothorax by means of the introduction of nitrogen gas into the pleural space, nearly every writer on the subject having detailed certain minor modifications in the technic. The technic, in a very general way, includes anesthetization of the skin, subcutaneous tissue and pleura by a deep injection of novocain, after sterilization of the skin with tincture of iodine, usually in the mid-axillary line at about the level of the fourth or fifth interspace, although at times it may be found necessary to make the injection further posteriorly, at a lower level. The skin is then incised with a sharp, thin-bladed (cataract) knife, and the blunt Floyd needle, connected with a water manometer, is inserted into the pleural space. After having secured a partial or complete collapse of the lung, in making the subsequent reinsufflations it may not be necessary to make an incision and use the blunt needle, the injections being made by puncturing with an ordinary aspirating needle. The greatest care must be exercised to avoid infecting the pleura, by using every precaution to perform the operation in a thoroughly aseptic manner. As soon as the entrance to the pleural space has been accomplished, a fluctuation in the water manometer is obtained, ranging between negative 2 cm. and negative 4 cm. Where numerous dense adhesions exist, the fluctuation may be very slight, or it may be impossible to obtain any fluctuation at all. No gas should be inserted unless the proper fluctuation has been obtained. When the adhesions cover only a moderate proportion of the pleural surface, repeated injections may be followed by the drawing out of the adhesions to form connective tissue bands transversing the pleural space, which eventually may permit the lung to collapse completely. The warm, filtered nitrogen gas or atmospheric air may be injected slowly at the rate of about



100 mils per minute until 500 or 600 mils have been introduced, provided that no symptoms arise to contraindicate the further injection of the gas, such as severe pain, cough, distress, sense of tightness, or shock; and on withdrawing the needle the opening in the skin should be sealed. It is advisable to control the injection by taking manometric readings after every 100 mils of gas have been injected, ceasing the introduction of the gas when slight positive pressure (4-6 mils) has been obtained, or 500 or 600 mils have been injected. In injecting the gas it is important to avoid using an excessive amount of pressure, and in the majority of cases no pressure

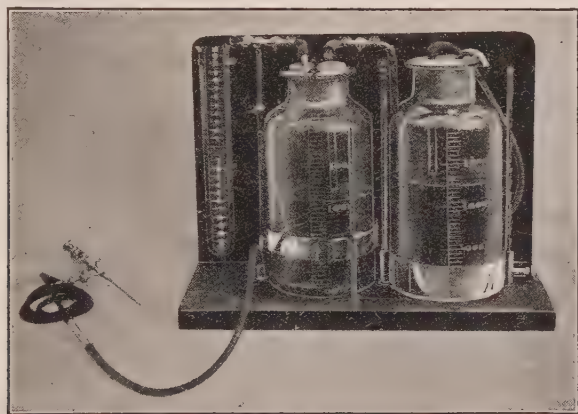


Fig. 18.—Apparatus for inducing artificial pneumothorax. Attached to the rear upright, on the reader's left, is the water manometer for determining the intrapleural pressure.

at all is required. Some writers recommend a larger quantity of gas when reinsufflation is performed, even up to 1000 mils at times being injected, these reinsufflations being recommended at first every other day, then twice a week, once a week, once in two weeks, until an effective collapse has been obtained, and then only once a month, or at even longer intervals. There seems to be no uniformity of opinion as to when the injections may be discontinued, but all agree on the importance of studying the cases carefully by means of frequent physical examinations and repeated skiagrams. The presence of fluid, when only slight in amount, seems to be most accurately determined by skiagraphy.

Nitrogen gas was employed originally in making the injections because it was believed that it was not so rapidly absorbed by the pleural surface. During more recent years sterile, warm atmospheric air is being more extensively used, as it tends to simplify the operation, and because it has been shown that there is very little difference between the diffusibility of nitrogen and atmospheric air.

Whenever suggesting the employment of the artificial pneumothorax to a patient, it is advisable to explain in a general way the theory upon which it is based, the chances of favorable results, and the advantages and disadvantages attending its use. Nothing could be more unwise than to urge the employment of such a method of treatment even in cases in which it seems to be specially indicated, without first supplying the patient with such information that it may be possible for him to decide for himself as to whether it should be used or not.

Some clinicians have attempted to secure an effect similar to that obtained by artificial pneumothorax by insisting upon the patients lying as much as possible on the affected side. Whenever possible, it would appear advantageous to encourage the patient to sleep upon the affected side in order thereby to limit the expansion of that side. The same conditions are secured by strapping the affected side of the chest, a method which may be employed with advantage in selected cases. Certain patients are unable to sleep on the affected side on account of the constant cough which attends any effort to maintain such a position. The straps are also exceedingly disturbing, on account of the sense of oppression which occasionally follows their use, and because of the irritation of the skin which may result from a prolonged application of the adhesive plaster.

#### SPECIFIC TREATMENT.

*Medicinal.* The medicinal treatment of pulmonary tuberculosis consists mainly of the correction of certain symptoms or complications, as the drugs which have been credited with specific value in the treatment of the tuberculous process have proven far from satisfactory. The majority of patients do best when the amount of medicine taken is kept down to the

smallest quantity possible. Numerous drugs from time to time have been lauded as specifics in this disease, but the fact that they have been discarded as useless serves to show how little real value they possessed. Arsenic, phosphorus, mercury, cinnamic acid, ichthyol, iodoform, menthol, eucalyptol, and numerous other drugs have been recommended in the past as of value in the treatment of this disease, but further experience proved their lack of specific value. It is to be hoped that some of the systematic investigations now under way ultimately will develop some combination of chemical agents which may prove efficacious in the treatment of this disease.

Before considering the treatment of the various symptoms and complications of this disease, it might be of interest to give some consideration to a drug which has been considered of benefit in the treatment of this disease, although in recent years it has been somewhat discredited.

*Alcohol.* The use of alcohol at one time was held to possess a value in treating tuberculosis which was considered almost specific. It is fortunate that the "barrel of whiskey and a barrel of cod-liver oil in the wilderness" method of treating the disease never received a very general acceptance over any considerable period of time. The effect of this teaching is still felt, however, and it is not an uncommon practice, even at the present time, to add alcohol to the dietary or medication of tuberculous cases. While the addition of alcohol in the form of whiskey may occasionally prove of value in the treatment of acute conditions arising during the course of the chronic forms of tuberculosis, it should always be combined with other drugs, being given as a medicine and never permitted as a beverage. The continued use of alcohol must be considered a very serious detriment in practically every case, and it should never be employed except to meet some distinct indication, and then discontinued as soon as possible.

There are several serious objections to the use of alcohol, aside from any deleterious effect it may or may not possess upon the resistance of the individual. In the first place, the use of alcohol by the patient himself in the early stages of the disease will not infrequently permit him to continue his ordinary occupation for a longer period than it would be pos-

sible for him to do without its use. Thus, not infrequently, the patient is finally obliged to discontinue his work and to come under a physician's care at a period when the disease has made such inroads as to be practically incurable, instead of at that early stage when the results of treatment are best. The abuse of alcohol is responsible in many instances for the patient's losing the possibility of an arrest of the process, with the functioning power of the lung almost fully preserved.

The objections to its use during treatment is that it gives a false sense of well-being and strength, usually resulting in overexertion and fatigue directly due to the fictitious feeling of strength created by this drug. The chief objection to its use is that in a chronic disease of this kind, where the treatment is almost certain to be long drawn out and tedious, we have need of all the moral stamina which the patient possesses to adhere to the rather rigid regulations necessary for an arrest of the process. The "moral anesthetic" effect of this drug, or its ability to destroy the will-power of the individual, must be considered its greatest and most serious drawback.

*Tuberculins and Sera.* Since the use of tuberculin was first suggested in the treatment of tuberculosis there have been innumerable preparations recommended, their claim for originality and virtue depending upon variations in the technic of manufacture, upon the source of the bacilli, the portion of the culture used, and similar details. The mere fact that so many different kinds of tuberculin have been recommended would indicate that the results following its employment have been far from satisfactory. It may be stated that at the present time there are very few experienced clinicians outside of large private sanatoria who employ tuberculin or recommend its use in the treatment of pulmonary tuberculosis. That it possesses distinct possibilities for harm is generally acknowledged, and every observer urges the necessity of extreme caution in its administration. While favorable results are occasionally reported from its use, especially in surgical and localized tuberculosis, the reports based upon its use in pulmonary tuberculosis, when viewed impartially, cannot be said to be very convincing when the results are compared with those obtained when it has not been em-

ployed, and the same statement can be made in regard to the various sera which have been recommended in the treatment of this disease.

The more one studies the various agents suggested as possessing value of a specific character in the treatment of tuberculosis, the more one is impressed with the importance of availing oneself of the various measures we possess for increasing the resistance of the individual by improving their general health. At the same time further study and investigation, both clinical and laboratory, should be directed toward the development of a therapeutic agent which will possess a definite specific action upon the tuberculous process, as it is impossible to estimate the enormous effect upon the general health of the community, with all its widespread influence upon economic and social conditions, which would result from the possession of an agent which would do for tuberculosis what salvarsan has done for syphilis. It has been proved beyond question that tuberculous subjects are especially susceptible to suggestion, and probably the psychic influence of tuberculin treatment is largely responsible for what favorable results have followed its use. A very serious objection to the use of tuberculin is that we possess no ready method for estimating the toxicity of any preparation, for even when made by the identical method they may vary greatly. One may readily see that it is impossible to lay down any fixed rules as to dosage or frequency of administration.

The numerous preparations of tuberculin fall into three large groups: (1) those composed of toxins produced by the tubercle bacilli in artificial culture media, like Koch's old tuberculin (O. T.); (2) those containing the tubercle bacilli themselves or their endotoxins, like Koch's bacillary emulsions (B. E.); and (3) a combination of (1) and (2), like T. R. As the effect of any tuberculin when injected into the body is apparently the same, it does not appear as if it made any difference which type of tuberculin was employed. It has been recommended that tuberculins of the type 1 of old tuberculin should be employed in those cases in which the evidence of general toxemia was out of all proportion to the degree of activity of the pulmonary lesion, as determined by physical examination, and type 2 tuberculins should be em-



ployed in those cases in which the pulmonary invasion was especially marked, with only relatively slight evidence of general toxemia; type 3 tuberculins being used in the cases in which the two processes were apparently equal.

The dilution of the tuberculin should be made with distilled water or 0.8 per cent. salt solution to which has been added 0.5 per cent. of carbolic acid, and it is advisable to use only freshly diluted tuberculin. For diluting the tuberculin one should have six to ten amber colored bottles, clean and sterile, labeled I, II, III, etc. In bottle I is placed 0.1 mil of tuberculin to be employed and 9.9 mils of the diluent. Each mil of this bottle will therefore contain 0.01 mil or 10 c.mm. of tuberculin. In each of the remaining bottles is placed 9 mils of diluent. One mil from bottle I is then placed in bottle II, each mil of which will then contain 1 c.mm. of tuberculin. When 1 mil of II solution is placed in bottle III, the latter will contain 0.1 c.mm. of tuberculin in each mil of solution. By continuing this process each mil of bottle IV will contain 0.01 c.mm. of tuberculin; of bottle V, 0.001 c.mm. tuberculin; of bottle VI 0.0001 c.mm. tuberculin; and each mil of bottle VII will contain 0.00001 c.mm. of tuberculin. This process may be continued until finally a dilution is reached where each mil will equal ten times the desired initial dose.

The initial dose will be 0.1 mil of the selected dilution, which may be continued indefinitely or gradually increased. In increasing the dose it has been suggested that it be not increased by 0.1 mil each time, as that doubles the original dose the first time it is increased, the proportionate increase in the dosage gradually diminishing as one approaches the full mil. It has been recommended that the dose be increased by  $\frac{1}{4}$  each time, which would give a dose increase approximately as follows: 0.1 mil, 0.12 mil, 0.15 mil, 0.19 mil, 0.24 mil, 0.30 mil, 0.37 mil, 0.46 mil, 0.57 mil, 0.71 mil, 0.89 mil, and 1.1 mil, or 0.11 of the bottle with the next lower number. It is advisable to keep the amount of fluid about 1 mil or less, in order to avoid injecting too large a quantity under the skin at one time. The injections may be made in the arm or back, the latter being the preferable location, but having the objection of being less accessible.

The initial doses recommended range anywhere between 1 mg. and 0.0000005 mg., the latter being preferable because least likely to do harm. Systemic reactions should be avoided by exercising great care in increasing the dose, but occasionally they appear to be unavoidable. In the event of a reaction, the succeeding dose should be well below the amount first given, and the same dose continued for some time before being increased, or the dose should be increased very gradually. The intervals between doses should be about two or three days in length, the treatment being continued until a point is reached where it appears desirable to discontinue for some reason or other. The various advocates of tuberculin are far from being unanimous in their opinion as to when the dose should not be further increased, or the treatment discontinued.

Tuberculin treatment should never be employed in the actively progressing forms of pulmonary tuberculosis, when there is evidence of marked activity, or in cases with distinct cardiac or renal disease. The least indication of increasing activity in the pulmonary process should be a warning that the administration of tuberculin should be discontinued.

#### DISPENSARY TREATMENT.

Tuberculosis is very prevalent among many persons who are unable to receive proper medical attention for economic reasons. In the large cities it is quite a problem to determine how these destitute individuals may be taken care of most efficiently and economically. It is generally recognized that sanatorium or hospital treatment is the best method for patients during the active and advanced stages of the disease (as discussed in the section on General Considerations, p. 406), and this leaves a large group for whom sanatorium and hospital treatment is either unnecessary or impossible. Those who are unable to pay for medical attendance and require home treatment are best managed by the dispensary service, for numerous reasons. In establishing dispensaries it is important that provision be made for clinics whose sole function shall be the treatment of tuberculosis, and that the dispensaries be so located that they will be easy of access, and that the districts covered by each dispensary will

not overlap. It is equally important that the dispensary confines its work to its immediate neighborhood, so that the home visitation can be most effectively performed.

The cases suitable for dispensary treatment are: (1) those who have had the disease sufficiently arrested to warrant their returning to work, but who still need to be kept under observation to prevent a recurrence; (2) that large group in whom the disease runs a very quiet, chronic course, which interferes very little with their general health; (3) children who show evidence of infection, and whose general health is so poor as to warrant fears that the disease may become active; (4) a small group in which the disease is active, but for various reasons it is deemed advisable to retain in their own home. With these groups of cases alone the dispensary should deal.

The dispensary which is fulfilling its duty in a thorough and efficient manner should not stop with merely treating patients, but should perform other equally important functions. The object of the dispensary should be the detection of the disease in its earliest stages, by attracting patients by every possible means, and by the examination of the other members of the family of every patient attending the dispensary, whenever this is possible. It should also act as a medium for educating the patients, their families, and their friends in the means of prevention of the disease and also in the value of ordinary hygiene and proper living. The hold upon the patient should also serve as a means of insisting upon their carrying out the necessary preventive measures, the visitation of the homes helping to insure that they are properly and thoroughly carried out. Finally, the work of the dispensary should be carefully and accurately recorded, so that the material collected may be available for analysis and study. The investigation of such important problems as are connected with housing, workshops, infection, and so forth should be an important part of the dispensary's paper work.

It is impossible to describe in detail the manner in which the work of prevention, education, and investigation can be best carried out, but the treatment of these dispensary cases is sufficiently important to warrant its being described at some length. For the first examination in all cases it is best to have a personal consultation between the physician and the

patient. The large proportion of the patients can be treated in a more satisfactory manner by what is known as the "class method" as evolved by Dr. Pratt. This method of treatment consists of holding conferences with the patients once every week or two weeks, at which a talk is given by the physician in charge upon various topics connected with the subject of tuberculosis. These talks should cover such subjects as rest and exercise, diet, symptoms, fresh air, prevention, infection, nature of the disease, etc., going into such detail as is necessary to give the patient a general knowledge of the subject discussed, so that he may co-operate with the physician and nurse in an intelligent manner. Each patient is provided with a book in which he keeps a record of the weight, temperature, and pulse, after instruction by the nurse; the food consumed daily, both as to character and amount; the number of hours spent in the open air; the daily amount and character of the work or exercise; any unusual symptoms or change in previous symptoms; and the treatment carried out. In this way a complete record is kept of the daily lives of the patients, which is much more accurate and reliable than that given from memory. These records are gone over carefully each week, and any mistakes are noted and used as texts for the talks given to the class. The patients are also given an opportunity to ask questions in regard to tuberculosis, the entire class receiving the benefit of the answer.

By this class method of treatment it is possible to develop the intelligent co-operation and *esprit de corps*, which could not be gained in any other way. It is also possible in this way to go into minute detail in regard to the purchasing, selection, preservation, and preparation of food, and the nutrient value of various articles of diet, which is impossible in the regular dispensary method. The patients can be shown how to make beds properly for sleeping in the open-air, and how to make their own window tents, home-made refrigerators and fireless cookers. They receive an education such as would equal that obtained in the best sanatoria, and, in addition, obtain information such as is provided by very few sanatoria. The mistakes of others serve as warnings to all, and the improvement of every patient acts as a stimulus to the others to do likewise. The results obtained by this method of

treatment naturally depend upon the enthusiasm, patience, tact, and resourcefulness of the physician and nurse in charge, and to a much greater extent than is ordinarily the case. Some care is necessary in the selection of the patients who seem adapted to this method of treatment.

Children who are below weight, anemic, subject to frequent colds, and who present evidence of infection may be treated by this same method, only modified to meet the altered conditions. The talks should be simple, and only such subjects covered as fresh air, overexercise, tea and coffee, excessive candy-eating, cleanliness, and care of the teeth,



Fig. 19.—Appliances used for instructing patients in the use of home-made refrigerators and fireless cookers. The charts shown on wall are used to illustrate the relative value of various foodstuffs. (Henry Phipps Institute, University of Pennsylvania.)

nails, and hair. The children may also be encouraged by prizes for improvement in health or general cleanliness, if deemed advisable. The weight, temperature, and pulse are taken only every two weeks, and no daily record is kept. After the talk has been given, the children are seen one at a time, and allowed to return home at once, unless there is special reason for an examination, such as excessive temperature, loss of weight, or increase of symptoms. While a few of the children may show signs of tuberculous disease, either of the glands, bones, or lungs, the majority of those suitable for this method of treatment belong to that large group in



which one suspects the possibility of beginning disease in an infected child, who betrays no definite evidence of tuberculous disease.

The proper treatment of dispensary patients is practically impossible without a certain amount of home visitation by experienced, tactful, well-trained nurses. By "well-trained nurses" is meant nurses who have had, in addition to their hospital training, instruction in public health and social service work. Hospital training is necessary for their proper appreciation of illness and all that it implies, and an experience in the treatment of disease. In no other way can a dis-

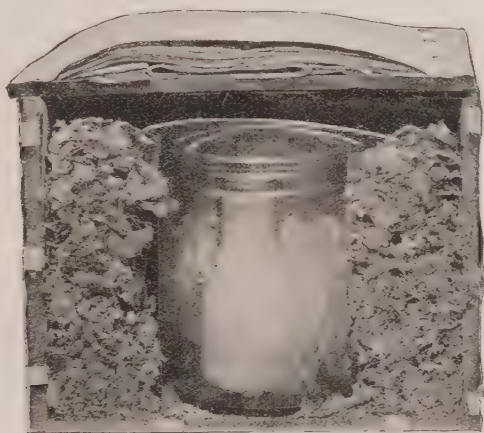


Fig. 20.—Cross section of home-made refrigerator. (Henry Phipps Institute, University of Pennsylvania.)

pensary worker gain a foothold in a family as adviser and friend save through actual assistance at the bedside of the sick. The public health work is also necessary to secure a broader view of the question of disease, the methods by which it may be studied, and its effect upon the general public welfare, together with the factors which are concerned in its cause and dissemination. A social service experience is of the utmost value, inasmuch as it helps to solve the economic factors which play such an important part in many cases, suggests plans for correcting the evils discovered, and provides ways and means for bringing to bear in the individual case the resources of the charitable and philanthropic organiza-

tions, or to assemble the family resources, as the case may be. The dispensary should not supply the patients with anything obtainable by any other means, so that it may not be viewed as a charity. A dispensary should be looked upon by the patients as a place where they may go freely, when unable to pay a physician, to receive advice and medical attention. This feeling can be secured only by careful attention, and not by the distribution of food and money. When the dispensary doles out charity it is attended only by such patients as are unscrupulous or devoid of self-respect, or by self-respecting patients when the disease has become advanced, their every cent having been spent before they would resort to charity. While every means should be employed to prevent the dispensary being abused by those able to pay, it is a great mistake, in dealing with a disease like tuberculosis, to draw the line too closely, as even after the disease has become quiescent and a certain amount of work is permissible, the patient should, if possible, possess a certain amount of money in reserve, so that he may not be forced to work beyond his strength.

One of the greatest difficulties in the proper management of the invalid poor is their tendency to drift, or to be sent from one dispensary to another. This may be overcome by having connected with the tuberculosis dispensary such additional clinics as frequently may be required. Thus every well equipped tuberculosis dispensary should have certain adjunct clinics for the treatment of nose and throat conditions, diseases of infancy, and, if possible, a dental clinic. A gynecological dispensary equipped to handle prenatal cases is of inestimable value. While an *x*-ray laboratory, especially equipped for the work, may at times be of use, the difficulties attending the securing of an operator of sufficient experience make it appear wiser in the majority of cases to have the work done in a convenient *x*-ray laboratory attached to a general hospital, if one be nearby.

There are several plans which have been suggested to meet the needs of the tuberculous patients in whom the disease has become arrested, or who are unable to attend a sanatorium. For one who is able to work, the night dispensary will prove a deeply appreciated help. The night-camp situated just outside the city, or on the roof of some large



Fig. 21.



Fig. 22.

Figs. 21 and 22.—Open-air school. Conducted by the Henry Phipps Institute, University of Pennsylvania, on the roof of the College Settlement, Philadelphia, showing the necessary equipment for cold weather.

building, may also be of value to this same class of cases, by providing a place where they may sleep out of doors under the best conditions they can obtain. For the patients who must be at rest for a large portion of the time the day-camp may be employed, preferably within easy reach of the patient's home. The river piers, roofs, and parks have been used for these day-camps.

There is a large group of children who show evidence of infection without any definite signs of actual tuberculous disease; they are underweight, poorly nourished, pale, and usually subject to coughs and colds. In other words, their appearance makes one suspect beginning disease without any demonstrable evidence upon which to base such a suspicion. If these children are to avoid the later development of disease and become useful members of the community, it is necessary that their general health and nutrition be brought up to the highest point possible. It is equally necessary that they receive an education and such training and care as their home life permits. To care for this group of cases open-air schools have been developed, and in the larger cities are being provided in an ever-increasing number. The pupils spend their entire school-hours in the open air, either on the roof, or in a room from which the window sashes have been removed. To meet the altered conditions, it is necessary that they be provided with proper clothing and extra covering. A warm cap, mittens, sweater, coat, bloomers, and sitting-out bag are indispensable in very cold weather (Fig. 21). The success attending open-air schools, not only in the freedom from disease and in the improvement in general health, but also in quickened mentality, must be seen to be appreciated. A fairly extensive experience with open-air schools makes one wonder why all schools are not conducted upon this same plan. The general improvement to be manifested in health and mentality, if all children were educated in the open air, would more than repay the slight inconvenience which this method might entail—to say nothing of the saving in coal.

Children suffering from active tuberculous disease should not be placed in open-air schools. They are just as much in need of rest as adults who have an active process. For this reason the word tuberculosis should not be associated with



open-air schools, for it tends to mislead and give a false impression to the children and their families, and to the public.

One of the most important functions, therefore, of the dispensary is the gathering in of all cases suggesting the possibility of tuberculosis, rejecting the non-tuberculous or unsuitable, determining the presence or absence of activity in the tuberculous, and outlining the appropriate course of treatment as seems best suited to each case. Those with active disease are sent to a sanatorium or hospital, or placed under the proper conditions at home, and those with evidence of infection without active disease merely instructed in the proper way of living, and cautioned against the conditions which might tend to stir up activity. The cases in which there is doubt as to whether the pulmonary process is active or not should also be instructed in the proper mode of living, and should be told to report at frequent intervals, so that they may be kept under observation. There can be no question that there is considerable room for education in regard to the difference in the requirements, depending upon whether the case is merely infected or actually has tuberculous disease, not only among social workers, nurses, and dispensary workers, but among physicians as well. Probably one of the most important functions of an efficient tuberculosis dispensary is the opportunity it offers for the education of medical students, nurses, and social workers. The ignorance of this most important disease which still exists is appalling, in spite of the widespread campaigns of education, for this instruction has been directed mainly toward the laity, with the professional classes almost ignored.

#### TREATMENT OF SPECIAL SYMPTOMS.

**Gastro-intestinal Disturbances.** In a very large proportion of tuberculosis cases the symptoms which call for active medicinal treatment are referable to some disturbances of the stomach or intestines, and in many instances these symptoms are the first indication of the disease, and not infrequently precede for a considerable time any symptom suggesting disease of the lungs. It is unfortunate that in a condition where recovery depends to such a marked degree upon the nutrition of the patient that these defects of digestion should so frequently develop.



The most common symptoms are those dependent upon the lack of gastric motility, and diminished secretion, so commonly present in tuberculosis, even in its early stages.

It is impossible to consider in a general article all of the numerous gastric symptoms which may arise in the course of this disease, but there are a few which occur so frequently, or are so serious in their nature, that they merit being considered in some detail.

*Loss of Appetite.* In the early stages of the infection loss of appetite responds quickly to the outdoor treatment, and when this symptom appears, the first step for its correction should consist in an investigation of the amount of fresh air the patient is obtaining. In the event of it being clearly shown that there is no deficiency of fresh air, it is necessary to determine whether the evacuation of the intestinal tract is regular and sufficient. It is only in the more advanced cases that it will be found necessary to resort to the use of nux vomica, gentian, and similar drugs before meals in order to stimulate the desire for food. It must be remembered that when the patient is taking milk and eggs at fairly frequent intervals it may be that the time between feedings is too brief, on account of the lack of gastric motility, and it may be found necessary to give the feedings at longer intervals. Hot water in the mornings, with or without the addition of sodium phosphate (not the effervescing), frequently has a very beneficial effect. Iodin has been recommended in the treatment of this symptom in the form of the tincture of iodine (U. S. P.) administered in milk or water either one-half hour before or during the meal, the dose being a few drops three times a day, gradually increased to 10 drops (0.6 mil), or even more. Creosote in small doses, when properly administered, will in many instances prove invaluable in stimulating the appetite (see p. 448).

*Vomiting.* While this symptom is not uncommonly observed in advanced cases, it may also be seen occasionally early in the disease. Patients suffering from severe cough, or when expectorating large quantities of tenacious mucus, very frequently have attacks of vomiting after the coughing spells; and this also is very prone to occur immediately after the ingestion of food. In such cases it is obviously the cough and expectoration which require medication, and not the stomach, although special care must be employed in the selection of the expectorants to be used.

A very common cause of gastric distress, nausea, and vomiting is the formation in the stomach of large, firm clots of casein immediately after the swallowing of milk, under which circumstances such measures should be employed as will serve to break up the clots, with the formation of finely divided particles of casein, rather than the retention of large firm masses. For this purpose it may occasionally be advisable to allow the patient to take a small quantity of crackers or toast with the milk, but whatever is taken should be well chewed and mixed with saliva before swallowing, and never soaked in the milk. Various other measures that may be tried are: Moderate doses of sodium bicarbonate or milk of magnesia before each feeding; lime water in generous doses with the milk; sodium citrate, two grains (0.13 Gm) to the ounce (32 mls) of milk; dilution of the milk with 1 part of Vichy to 2 of milk; or possibly some of the commercial infant foods may be employed.

When vomiting of this type occurs, it is advisable to investigate carefully the diet, to determine whether too much food is being taken, or whether the intervals between the feedings are too short. It may be found that the patient is drinking the milk too rapidly, and it may be necessary to instruct the patient to drink the milk slowly, allowing a short pause between mouthfuls. Carefully instructing the patient in regard to the quantity, time of taking, and proper method of drinking milk, will very frequently avoid the necessity of administering medicine or modifying the milk.

Another type of vomiting occurs about one hour after eating, and is usually preceded by considerable distress, nausea, and eructations of gas. This type is also more commonly seen in advanced cases, but occasionally may be found fairly early in the course of the disease. This type of emesis requires the most careful treatment, and at times it may even be found necessary to discontinue all food by the mouth, and to resort to rectal feeding for a short period.

One of the best methods of handling such cases is to reduce the quantity of food to the minimum amount possible. No solid food should be taken, and the milk, only in small quantities, preferably peptonized, may be given at intervals of two and one-half hours. It is usually possible after a few days of such feeding cautiously to increase the amount of milk, and then gradually to

add other articles of diet. Certain of these cases respond very well to a decrease in the amount of fats, a few days upon skimmed milk and the white of eggs sufficing to remedy the digestive disturbance,

While the above suggestions are made in regard to vomiting, they apply equally well to some of the digestive disturbances which may occur without vomiting. A not infrequent cause of digestive disturbance is the swallowing of the sputum, which the patient may do unconsciously, and it is a good plan always to caution the patient to guard against its occurrence. There is nothing peculiar about the gastric symptoms incident to pulmonary tuberculosis, for they have the same significance, and call for the same methods of treatment, as when encountered in otherwise healthy individuals. A word of caution is not out of place in regard to the use of the stomach-tube. In an early case, in which the swallowing of the tube is not accompanied by any marked disturbance, such as violent gagging and retching, there can be no possible objection to its use. In an advanced case, subject to hemoptysis, or in an early case in which the posterior pharynx is very irritable, and in which the passage of the tube causes considerable gagging, it should never be employed unless absolutely necessary, and then only with the greatest care.

There are very few cases that will be able to take sufficient nourishment, especially when on complete rest, without being markedly benefited at some time during their course of treatment by the occasional administration of pepsin, dilute hydrochloric acid, nux vomica, sodium bicarbonate, charcoal, or creosote, according to the indications.

*Constipation.* When taking milk and eggs it is extremely important that the patient secures a prompt evacuation of the unabsorbed residue, as otherwise gastric and intestinal disturbances and evidences of general autointoxication will quickly manifest themselves. A very common belief among the laity is that milk is constipating, some refusing to take it on that account alone. While some do suffer at first from this distressing symptom, it can frequently be overcome by increasing the amount of milk taken. If this method fails to bring about the desired result, the usual methods of correcting constipation may be instituted, such as massage, a generous diet of coarse grains and fruit, at the mid-day meal, with vegetables, especially those of a type which supply

considerable fibrous residue and provide sufficient bulk to stimulate peristalsis. If it is necessary to resort to drugs, heavy mineral oil is by far the most satisfactory, although it may be necessary to use cascara sagrada in some instances. Phenolphthalein in doses of 3 to 5 grains (0.19 to 0.32 Gm.) will occasionally be found useful. The occasional administration of a saline purge, preferably magnesium sulphate, is almost essential in patients in whom the feeding is being forced, especially when they are on absolute rest. The occasional use of calomel, always being followed by an active saline purge, seems to be of considerable benefit in some instances.

*Diarrhea* is a symptom which occasionally develops in patients on a milk-and-egg diet, or even at times when one is taking three regular meals a day. In acute attacks, thorough evacuation of the bowel with Epsom salts or castor oil, with food totally suspended for from six to twelve hours, and then cautiously resumed in the form of a bland, non-irritating diet, will usually prove sufficient. If, on the other hand, it should persist, the general digestive function of the patient should be carefully studied, and an effort made to discover just what is responsible for the loose bowels. It may mean incomplete digestion, with the appearance in the stools of large quantities of partly digested food; occasionally the fats may be responsible, or possibly it may result from an unsuspected constipation with resulting irritation and fermentation. A daily movement of the bowels does not exclude the possibility of constipation, since loose material may tunnel through a tenacious mural mass of fecal matter. One should always investigate the purity of the milk, as in many instances contaminated milk is responsible, but in some cases it will be found that the only cause for its appearance is the milk itself, even when perfectly clean and pure. One should discontinue milk as a food only after being thoroughly convinced that it is responsible for the symptoms, by a careful study of the gastric digestion and the correction of any defects found. A trial of the various intestinal ferments or of the agents used to stimulate their secretion should be made, and a careful investigation of the source of the milk supply, with, if necessary, a bacterial count of the milk, is to be undertaken; and the fats eliminated for a short period, substituting skimmed for the whole-milk. If the milk is not found responsible for the diarrhea, and if this symptom



should continue even after it has been discontinued, decrease of the starchy and fatty food for a short period, with a relative increase of the proteids, and the administration of bismuth subnitrate, bismuth subgallate, or one of the tannin preparations, frequently corrects the condition. Where considerable fermentation accompanies the diarrhea, active cultures of Bulgarian bacilli, small quantities of heavy mineral oil, charcoal, and creosote will be found of considerable value, and in some cases an abdominal binder may relieve the tendency to frequent watery movements.

In advanced cases it has been shown by *post-mortem* examinations that tuberculous ulceration of the intestines is of very frequent occurrence, and in the presence of persistent diarrhea this factor should be carefully considered as a possible source of this distressing and grave symptom.

In treating any and all of the disturbances of digestion one must constantly remember that the disorder may not be due to any organic condition, but may be essentially of nervous origin. Many of the disturbances will frequently respond to moderate doses of bromids, which plan of treatment should be given a trial before resorting to more radical measures in any case in which there is a probability of the disorder being of this type.

**Cough.** The cough accompanying tuberculosis may be due to a variety of causes, and in the treatment of this symptom it is necessary to study the patient with the object of determining its sources before resorting to medication. Laryngeal implication or inflammation, abnormalities of the upper air-passages, digestive disturbance, pleural effusion, enlarged bronchial glands, or pulmonary congestion may be responsible for the cough. As a matter of fact, in the properly managed case of pulmonary tuberculosis, cough is not a very prominent or distressing symptom, notwithstanding the view to the contrary so commonly held. A patient who is under the proper general conditions usually coughs only when there is a collection of mucus to be expectorated, and the cough necessary to raise the mucus is usually so slight as to be hardly noticeable. The patient with pulmonary tuberculosis is usually given entirely too many drugs, and most of these are directed toward checking the cough. So long as there is a tendency toward activity of the process in the lungs, there is bound to be a certain amount of expectoration, which is really one of nature's methods of elimination. It must be evident, therefore,



that the expectoration of a moderate amount of material is necessary for the welfare of the patient, as evidenced by the rise of temperature and other evidences of toxemia so frequently appearing upon the cessation of expectoration, or a sudden decrease of its amount. It is only when the expectoration is accompanied by severe coughing efforts that any medication is required, or when there is a very hard, dry, unproductive cough. Many patients who complain of considerable unproductive cough may be taught to suppress it. It is astonishing how much can be done with some patients along this line, when the physician's instructions are faithfully followed. In a properly conducted sanatorium, whose inmates have instilled into them constantly the importance of not giving way to the desire to cough, even when many of them are advanced cases, it is remarkable how little coughing one hears, except possibly in the early morning hours. It is important to impress upon the patient the fact that one cough leads to another, and that the effort to suppress a cough assures them of a considerable period without this symptom. They may be encouraged by being informed that a few days of effort are all that is necessary, as after that time the suppression becomes automatic. The morning cough is necessary to remove the mucus which has collected during the night, and does not require any special treatment, unless the mucus is of such a tenacious character that too violent an effort is required to dislodge it.

Tuberculous patients, like every one else, at times may develop a cough which will require treatment, but many of them will be saved a great deal of discomfort, if not actual harm, if the attending physician would only bear in mind that cough is a symptom in pulmonary tuberculosis which does not usually require medical treatment. The acute or chronic bronchitis in a tuberculous patient requires the same treatment, according to indications, as other individuals, as described in the chapters dealing with these conditions. (See *Acute and Chronic Bronchitis*, p. 326, *et seq.*)

The question of deciding whether a patient may be permitted to smoke must be decided in the individual case. The factors influencing one in deciding that smoking must be discontinued are the presence of pharyngeal or laryngeal inflammation, failure to gain in weight, gastro-intestinal disturbances, or persistent unproductive cough. Many patients who have smoked for years derive an immense amount of comfort from tobacco, without the least

apparent harm in many instances. Excessive smoking should be absolutely forbidden. The period immediately after eating seems to be the one in which the craving for tobacco is greatest, and the time during which it is most advisable to permit its use. Inhalation of the smoke should be discouraged, and for this reason the use of cigarettes should be forbidden, as one finds it easier to refrain from inhaling the smoke from a pipe or a mild cigar. The use of the pipe is probably best in these cases, as it permits of a wide range of tobacco from which to select, and no tobacco or its products enters the mouth, provided the stem of the pipe is frequently cleansed. When the habit is not of many years standing, or when the patient does not progress favorably, it is probably safer to break off smoking entirely than to attempt to limit the amount.

Creosote is one of the drugs which has been credited with special and peculiar virtue in the treatment of pulmonary tuberculosis, and it might be of interest to say a few words in regard to the indications for its use, the method of administration, and other details.

At the time creosote was first recommended, the only form in which tuberculosis of the lungs was recognized was what is termed consumption—in other words, only in the advanced stage or actively spreading form. Those of the hectic type, or what is now considered as those with mixed infection, were the cases in which this drug was considered as being especially indicated. It is in just these cases that creosote gives the best results, when the expectoration is profuse and purulent, and when there is evidence of a breaking-down process in a consolidated or densely infiltrated pulmonary focus. It is not indicated in the average early or moderately advanced patient, in whom these conditions are not usually present. There is a common belief that creosote tends to disturb digestion, and should not be employed where there is any sign of gastric disturbance. On the contrary, when properly given, it may be continued for long periods of time, not only without interfering with digestion, but in many instances improving the appetite, and correcting any tendency toward fermentation in the gastrointestinal tract. The method of administration which seems to have given the best results in the hands of the writer is in moderately small doses, one drop (0.07 mil) gradually increased to 5 (0.35 mil) or even 10 drops (0.64 mil), stirred in a large cup

of very hot water, and not being drunk until the small droplets can no longer be seen floating upon the surface. The best time to give it is one hour before meals, but as this is very difficult to carry out before breakfast, it is usually more convenient to administer it only twice daily, preceding the mid-day and evening feedings. While many advocate the employment of larger doses, the amount stated usually will be found sufficient, and better results generally are obtained if the drug is administered for a few weeks, then discontinued for a week, and then renewed, these short periods without the drug being of considerable help, when it is desirable to continue the use of the creosote over an extended period of time.

Opium derivatives, to control the cough, are employed entirely too frequently in pulmonary tuberculosis. They are of considerable value in exceptional cases, or in certain complications, but should never be used as a routine measure in early or in moderately advanced cases. When one is dealing with an advanced case, on the contrary, it is criminal to withhold the comfort and relief which may be obtained from heroin, codein, or even morphin. The unfavorable after-effects are more than counter-balanced in these instances by the prolonged periods of relative comfort, and there is no danger of forming a habit which might prove as serious as the original disease, as in the case of those in whom there is a possibility of recovery from the tuberculous process.

When the mucus is very tenacious, and accompanied by hard coughing spells, there is no drug which can be compared with ammonium chlorid in 5-grain (0.32 Gm.) doses four to six times a day, preferably after meals. This drug may be continued for months, if so desired, without interfering in any way with digestion, provided that it is given in an aqueous solution, or with one of the bitter elixirs, such as the elixir of calisaya. It should never be given in the various popular syrups, as the patient will soon be forced to discontinue its use on account of gastric disturbance. These cough-syrups are undoubtedly responsible for the prevalent belief that ammonium chlorid is a drug which should not be continued for any length of time on account of its deleterious action upon the stomach.

**Pain.** There are very few cases of pulmonary tuberculosis that go through the course of their disease without some pain in the chest at some time or other. In considering the pains occur-

ring in this disease, no further reference will be made here to the pains due to a frank pleurisy or to gastric disturbance, as they will be considered under the chapters dealing with those subjects.

A very common type of pain is the one referred to the region of the chest between the scapulæ, or in the area corresponding to the angle of either scapula. This pain usually arises when patients have permitted themselves to become exhausted, either mentally or physically, and is of a dull, boring, neuralgic character. The remedy for this type of pain consists of absolute or approximately absolute rest, and the building up of the patient's general strength. Local remedies occasionally will give relief, especially counter-irritation with tincture of iodine or mustard, or in some cases the salicylates may give temporary relief, although there are numerous objections to their being continued for any considerable time.

Pain in the shoulder is not at all uncommon, and is usually extremely difficult to relieve; the severe, sharp pains which usually occur in the shoulder on the affected side, but may affect the opposite shoulder, are usually worse at night. Dry heat is the measure which usually gives the greatest amount of relief, combined with rest of the joint and gentle massage or counter-irritation with iodine, or small blisters, frequently repeated. The pains may occur in other joints in the body, such as hips, knees, elbows, or wrists, and when not due to definite disease of the joints, may be treated along the same lines as suggested for the management of the pains in the shoulders. It is advisable in these cases to search for hidden foci of infection, including a careful examination of the teeth.

A dull, aching pain, or occasional sharp, shooting pains, occur fairly frequently over the upper anterior chest. It has been thought that these are produced by pleural inflammation or adhesions, which usually accompany an apical process. While the pleuræ may be responsible in some cases, this cannot always be true, as the pains not infrequently occur upon the unaffected side. It would seem that certain of these pains must be muscular, or possibly neuralgic, on account of their location and general character. The same local and general measures may be employed in this type of pain as that previously described. Very often dry cups, or small blisters, frequently repeated, prove of benefit. As the drugs which may be used for the relief of these pains are certainly not beneficial, if not actually harmful, to the general con-



dition of the patient, it is desirable, whenever possible, to employ local measures for their relief, rather than internal medication.

It is a very good practice to warn patients that these pains are likely to occur, that they do not signify inflammation of the lung, or extension of the disease, and to allow them to paint the chest with tincture of iodine when they occur. Another convenient method of securing counter-irritation is to have the patient soak a few rough towels in a strong salt solution, which are then allowed to dry, and may then be employed for rubbing the chest when the pains occur. While heat will at times relieve the pain, most patients seem better off if they can secure the desired relief by the other local measures above enumerated.

Rubbing the chest with oil seems to be followed by more relief in some cases than can be obtained by any other measure, especially when a certain proportion of oil of gaultheria is added. When it is desired to administer iodine, some of the various preparations may be combined with the oil. A favorite formula for rubbing with oil, highly recommended by some physicians, not only for the relief of pain, but for its apparent beneficial effect upon the general condition, is the following:

Oil of gaultheria ..... f3ij (8 mls).  
 Euophen (28% iodine) ..... 3ij (8 Gms.).  
 Olive oil (or cottonseed oil)  
 q. s. ....ad f3vj (64 mls).

Sig. One teaspoonful (4 mls) rubbed into the chest night and morning.

For the same purpose the following mixture is applied in the same manner:

Iodin crystals ..... gr. xxx (2 Gms.).  
 Lanolin ..... 3ss (32 Gms.).  
 Olive or cotton-seed oil..q.s.ad 3vj (192 mls).  
 Ether ..... q. s.

**Hemoptysis.** It has been said that 60 per cent. of tuberculous cases present this symptom at some time during the course of the disease. The degree of the hemoptysis may vary from blood-streaked sputum to the sudden massive hemorrhages which cause death almost instantly. There must be many causes for the raising of the blood, the slight bleedings being probably due to congestion or the rupture of small vessels, while the raising of small clots, so frequently seen in cavity



cases, is almost certainly due to oozing from small vessels. In others this symptom is accompanied by signs which seem to indicate the presence of a pneumonic process, possibly as a result of the rapid extension of the tuberculous disease. There are certain cases in which the hemorrhages do not occur until the patient is apparently well on the road to recovery.

From this exceedingly brief description can be readily seen the difficulties which attend any effort to lay down a fixed rule or outline a single method of treating this symptom. In treating any case of pulmonary hemorrhage an effort should be made first to discover the exact cause of the bleeding. While theoretically this is extremely desirable, in practice it presents many difficulties, the greatest of which is the danger attending moving the patient to the extent necessary for a thorough examination, and of the lungs particularly, with reference to percussion, and to the deep-breathing so necessary for a satisfactory auscultatory examination. This means that the source or nature of the bleeding must be determined largely by the temperature, symptoms, character of expectorated blood, and previous knowledge of the case, supplemented by what one may elicit by auscultation of the chest without change of posture or deep-breathing on the part of the patient. While this materially limits the field of investigation, one frequently may derive sufficient information upon which to base a very accurate conception of the location and nature of the process responsible for the bleeding. Many of the hemorrhages met with are not of a serious nature, being checked by very little or no treatment; but it seems hardly wise to proceed upon such an assumption, in view of the fact that some of them are so extremely serious. As our present means of differentiating the hemoptysis due to the various causes is not as perfect as one might wish, it would seem advisable to treat all of them, no matter how slight they might be, as a serious symptom.

A very important factor, if not the most important, in treating any case of hemoptysis, is for the physician to reassure the patient. The mental excitement and worry resulting from the expectorating of blood is usually extreme, and undoubtedly is frequently responsible for the continuation

of the bleeding, so that the most essential element in the treatment of the condition is rest, not only absolute physical rest, but mental as well. The patient should be put to bed, and not allowed to leave it for any reason so long as the bleeding continues, and in fact should be kept as near absolute quiet as is possible. The posture which the patient should assume is the one in which he is most comfortable and the one least likely to excite a cough. When the bleeding comes from a ruptured vessel, the most essential point in the treatment is the lowering of the blood-pressure, and absolute mental and physical rest are two extremely valuable means we possess for bringing about this reduction. The common practice of inducing quiet by giving repeated doses of morphin in all cases of hemoptysis is not to be recommended, for this drug possesses too many deleterious effects. The excitability and restlessness may usually be effectually controlled by moderate doses of bromids, but in some cases it will be found absolutely necessary to administer morphin or heroin, although never for any length of time, and only in amount sufficient to quiet the patient. In employing morphin one must be extremely careful not to give it in such doses as will produce heavy sleep or absolutely prevent cough. A certain amount of cough is necessary to remove the blood accumulating in the bronchi, in spite of any ill effects which may possibly result from the cough by disturbing the clotting at the bleeding-point. For further reducing the blood-pressure there is no drug which is as satisfactory as the nitrites, given in the form of nitroglycerin, amyl nitrite, or sodium nitrite. A tablet of  $\frac{1}{100}$  of a grain (0.00064 Gm.) of nitroglycerin, dissolved on the tongue, usually has the desired effect, and may be repeated sufficiently often to maintain the reduction of the blood-pressure. As it has been shown that tablets of nitroglycerin, are occasionally inert, the spirit of glonoin (1 per cent. alcoholic solution of nitroglycerin) may be employed in doses of 1 to 2 minims (0.0648 to 0.1296 Gm.) every hour. Some care must be exercised in employing the spirit of glonoin, as, if the preparation is stale, it may be stronger than it should be, as a result of concentration from evaporation of the alcohol. Owing to this tendency, the drug should never be prescribed in full strength, but should be diluted with water,

1 or 2 minims (0.062 or 0.123 mil) to the dram (4 mils) as desired. The nitroglycerin should be administered at frequent intervals, as its effect is very fleeting, the dose desired being repeated every hour until the pulse or the patient's symptoms indicate that the desired effect or physiologic limit has been reached, when the intervals between the doses may be increased. When a very quick effect is desired, especially in an emergency, the inhalations of amyl nitrite are to be preferred. *Veratrum viride* has also been recommended for this purpose, as it is claimed that lowered pressure is maintained for a longer period of time than is the case with the nitrites, in which the effect is very evanescent. A very common practice is to apply an ice-bag to the chest, over the bleeding-point when it can be located. As the ice-bag frequently affords a sense of reassurance to the patient, and helps to maintain the patient absolutely quiet, it may be continued if so desired, but preferably it should be placed over the heart, and not over the suspected bleeding-point in the lungs. The maintaining of a low pressure is very important, and for this reason all cases of hemorrhage should be carefully watched. The drugs which have been used as a means of reducing the blood-tension should never be suddenly withdrawn, but should be given at progressively longer intervals after the bleeding has ceased, and then discontinued at the end of several days. Emetin has also been used with considerable success in the treatment of hemoptysis, numerous very favorable reports having been published in the past few years upon the use of this drug. Emetin may be given, preferably hypodermically, in  $\frac{3}{4}$ -grain (0.0486 Gm.) doses three or four times a day, using either the hypodermic tablets or ampoules. The drugs which have been recommended at one time or another for the treatment of this condition are without number, but the majority of cases will respond better to the plan of treatment above outlined than to any other, as the use of numerous different drugs in this emergency frequently is responsible for unfavorable results.

In order to secure the formation of a clot at the bleeding-point, which is the object of all treatment of hemorrhage, there are two factors necessary: the tension of the blood must be reduced, so that the clot may not be forced away from

the opening, and the blood must possess the ability to coagulate. Unfortunately the methods at our command for increasing the coagulability of the blood are either unreliable or do not act quickly enough to be of use in an emergency. For patients who are subject to hemorrhage, the administration of calcium lactate in full doses over a rather prolonged period of time may prove of benefit. Ordinary table salt has been for many years a very popular home remedy for hemoptysis and may be employed in 5- to 15- grain doses (0.324 to 0.972 Gm.). It has been shown that sodium chlorid increases the coagulability of the blood very quickly, but the effect is not lasting. Ten to 15 mils (2 f3 42 min. to 4 f3 4 min.) of a 10 per cent. solution of sodium chlorid, injected intravenously, also has been recommended, the solution being heated to body temperature before use. Care must be taken to prevent any of the solution leaking into the subcutaneous tissues, as it may cause extreme pain. Sodium bromid has very much the same effect as sodium chlorid, and is to be preferred for administration by the mouth, especially in nervous subjects.

The tying-off of the blood in the extremities by means of tourniquets has also been recommended for increasing the coagulability of the blood. The constriction of the arms or hips must be performed carefully, to avoid injury to the tissues, and should not be maintained longer than one-half to one hour, the bandages being removed very slowly at the end of this time. It is to be hoped that the more recent studies upon the coagulability of the blood will result in providing some rapid method for determining the factor at fault in any case with decreased coagulability, and will suggest some agent for quickly and satisfactorily supplying the deficiency. The rather empiric method for increasing the coagulability by injecting horse serum has been recommended, naturally with greatly varying results. Care must be exercised when horse serum is given repeatedly, to make sure that the patient has not become sensitized, and warning is to be given of the potential dangers, akin to those which may attend the administration of diphtheria antitoxin.

When the site of the bleeding can be absolutely determined, artificial pneumothorax has been strongly recom-



mended as a means of controlling the hemorrhages. This procedure has the disadvantage, first, of being applicable only to a limited number of cases, and, second, the disturbance of the patient necessary to carry out the procedure is not without a certain risk. Where the hemorrhages are severe and prolonged, and the bleeding-point can be located without any question, it offers a means of absolutely controlling pulmonary hemorrhage.

**Pyrexia.** There is nothing peculiar about the temperature course in pulmonary tuberculosis, unless one can consider its protean character as distinctive. The temperature in an uncomplicated case is practically always subnormal in the morning, although occasionally the inverse type is met with, in which the subnormal phase occurs in the afternoon. When the temperature continues elevated, it usually indicates the presence of some complication. A rise of temperature always demands rest in bed and careful observation, regardless of its cause, and it frequently indicates an increased activity in the pulmonary process. Slight temporary rises may result from over-exertion or fatigue, menstruation, over-excitement, gastro-intestinal disturbances, or the presence of an acute infection, such as coryza, tonsillitis, or bronchitis. The more serious conditions in which the first indication of their presence may be a rise of temperature are: pleurisy, hemoptysis, meningitis, pneumonia, and an acute general miliary tuberculosis.

Fever is not infrequent in pulmonary tuberculosis, and may occur at any stage of the disease, but is naturally more frequent, excessive, and resistant to treatment in the advanced cases. In the earlier stages, when due to an exacerbation of the pulmonary process, it will usually respond quickly to general hygienic measures, the most important of which is absolute rest, as described in dealing with the subjects of Rest and Exercise (see p. 412). Occasionally cases will be encountered in which these general measures appear to be insufficient to overcome the toxemia responsible for this symptom, and under such circumstances it will be found necessary to resort to other measures in order to make the patient comfortable. Bathing with water is by far the most satisfactory means of securing a reduction of the temperature. This may



be carried out with tepid, cool, or even iced water, depending upon the amount of fever and the rapidity with which the temperature falls after the bath. Some patients with even a fairly high fever respond quickly to the tepid bath, but in others iced water is required before the temperature tends to decline and a certain amount of comfort is obtained. The addition of alcohol to the water is very refreshing and agreeable to most patients. Occasionally a hot foot-bath will serve the same purpose as the cool bath, especially if mustard be added to the hot water.

In applying the cool sponge it will usually be found sufficient to bathe the extremities, although there is no objection to bathing the entire body, provided that care is taken to see that the patient is thoroughly dried afterward, without too brisk a rubbing with the towel.

Drugs for the reduction of temperature should never be resorted to, except in extreme cases. The class of drugs which will help to lower the fever has a distinctly unfavorable effect upon the patient, especially if persisted in for a considerable time. Occasionally, when a sudden rise of temperature does not respond to the measures suggested, and is accompanied by general severe discomfort and distress, it may be permissible to employ drugs for this purpose. The most satisfactory is phenacetin in doses of 2 or 3 grains (0.13 or 0.19 Gm.), repeated every two hours for three or four doses, in which quantity it may be considered safe and at the same time effective. Aspirin also may be given for this purpose in 5-grain (0.32 Gm.) doses three or four times a day. While quinin is without the depressing effect of some of the other antipyretics, it is seldom that it exerts any appreciable effect, but it may be tried when other methods have failed.

**Night-sweats.** A symptom which may prove very disturbing to the patient is the occurrence of profuse sweating at night. This is not only annoying to the patient, but usually leaves them with a sense of exhaustion, and, what is more serious, while in the wet state exposes them to chilling, which may have a very unfavorable effect upon their general condition. This symptom is of relatively infrequent occurrence in early cases, being more common in patients with moderate dissemination of an acute, actively spreading type, and in those with

extensive consolidation. As a rule, night-sweats occur in those patients who present that group of symptoms commonly believed to indicate the presence of mixed infection. Contrary to the belief commonly held, the sense of depression following the sweats is due to the conditions responsible for this symptom, and not to the sweating itself.

It will be found in nearly every instance that the mere placing of the patient under improved hygienic conditions, with sufficient fresh air, and a regulation of the diet, entirely checks the occurrence of this symptom. Whenever it appears, a careful investigation should be made of the patient's mode of living, inquiring particularly into the amount of rest, time spent in the open air, method of securing fresh air, clothing, bathing, diet, occurrence of constipation, and any other detail which suggests itself in causal relationship. It will usually be found that there is some hygienic error responsible for the night-sweats, the correction of which will prevent their further occurrence without any special treatment.

Cases are occasionally met with which require more active and specific treatment, especially those with a lesion of the caseating, actively spreading type. Here it will be necessary to try to improve the general tone of the patient by cool baths; these, when administered in the evening just before going to sleep, seem to be especially valuable. The addition of vinegar or alcohol to the cool water with which the patient is sponged occasionally increases its efficiency. A cool sponge in the morning, or a cold chest-bath, will often prove of benefit in these cases. It has also been advised to administer a small quantity of brandy or whiskey on retiring, or a short time before the sweats usually occur.

A glass of hot milk, or even a small quantity of solid food, such as toast or roll, may be employed instead of the alcoholic beverage. When the measures suggested fail to check the night-sweats, a trial may be made of various drugs, among which atropin sulphate, in doses of  $\frac{1}{100}$  to  $\frac{1}{75}$  of a grain (0.00064 to 0.00086 Gm.) on retiring, is by far the most satisfactory. Other drugs which have been recommended are: camphoric acid, calcium salts, and agaricin, which may be given a trial if other methods of treatment should prove unavailing, although it is only reasonable to expect very little from the

use of drugs in those cases in which the general hygienic measures prove useless. The ideal treatment for this condition naturally involves the elimination or correction of the toxins responsible for the sweating, rather than the mere relief of the symptoms.

**Anemia.** Anemia of the chlorotic type, of varying severity, is frequently present in pulmonary tuberculosis, and, while not infrequently present in early cases, is more common in the advanced.

This chloroanemia is of the type which one would naturally expect should respond to iron medication most readily, and yet in many instances the results are far from satisfactory. There are various forms in which the iron may be administered, such as the tincture of the chlorid of iron, Bland's pills (carbonate), peptomanganate of iron, syrup of the iodid of iron, and ovoid ferrin. The hypodermic administration of the citrate of iron is strongly recommended by some writers, and while this mode of administration possesses certain disadvantages the possibility of gastric derangement and constipation are eliminated. The following formulas have been recommended:

1. Iron citrate ..... 0.1      Gm. ( $1\frac{1}{2}$  gr.).
2. Iron citrate ..... 0.05      Gm. ( $\frac{3}{4}$  gr.).  
    Strychnin sulphate ..... 0.0005 Gm. ( $\frac{1}{120}$  gr.).  
    Sodium arsenate ..... 0.001    Gm. ( $\frac{1}{60}$  gr.).
3. Iron citrate ..... 0.05      Gm. ( $\frac{3}{4}$  gr.).  
    Metarsinate of sodium ..... 0.01    Gm. ( $\frac{1}{6}$  gr.).  
    Strychnin sulphate ..... 0.001    Gm. ( $\frac{1}{60}$  gr.).

These combinations are strongly recommended by Peters and Bullock, who state that the hemoglobin content of the blood may be brought up to normal by 20 daily injections.

In view of the unfavorable effect of arsenic in so many cases, and the questionable value which it possesses in the secondary anemia of pulmonary tuberculosis, it would seem to be much wiser to omit the use of this drug, unless great care can be exercised in its administration, and the patient kept under constant observation.

The best results in the treatment of this symptom in the average case are obtained by a generous supply of fresh air and nutritious food.

## COMPLICATIONS.

**Tuberculous Pleurisy.** The treatment of this condition has been covered in a separate section (see Pleurisy, p. 502), and yet it would seem advisable to call attention to several points in the treatment of special significance when this complication occurs in tuberculosis.

In the first place, the pleural effusion which occurs without any evident etiologic factor should be looked upon in the same light as one would view hemoptysis under the same conditions, namely, as in all probability being tuberculous in origin. The tuberculous pleurisy may precede distinct clinical evidence of pulmonary tuberculosis by many years, and should always be treated as any other case of tuberculosis regardless of the absence of pulmonary signs. Every case in which no evident cause for the appearance of an effusion can be discovered should be treated just as one would treat an incipient case of pulmonary tuberculosis. The patient should be brought up to the highest degree of nutrition, and not allowed to return to an ordinary mode of living until one has become convinced of their full ability to resume such a life. They should be constantly kept under observation, and a careful examination of the lungs made at fairly frequent intervals. It is best to insist upon a rational mode of life, with fresh air in abundance whenever possible, even after the patient has resumed his or her regular occupation. If after the pleurisy has cleared up, the patient is underweight, tires easily, or shows a tendency to an elevation of temperature or pulse, a period of treatment under careful observation such as one would obtain in a sanatorium is absolutely necessary to prevent the development of pulmonary tuberculosis at a later date.

*Treatment.* There is one point in regard to the treatment of pleural effusion in subjects of pulmonary tuberculosis which cannot be too frequently repeated, and that is the serious results which may follow the sudden withdrawal of large quantities of serum. Where there is tuberculosis of the lung, one of the most important points in the treatment is the obtaining of as nearly complete functional rest of the lung as possible. When a pleural effusion occurs there is produced naturally the conditions which we strive to obtain by artificial pneumothorax in the treatment of this condition—the lung is put at absolute rest. The sudden withdrawal of the fluid



causes abrupt distension of the lung, with not infrequently a rapid extension of the disease as a result. When the effusion becomes so massive that the mere bulk of the serum causes distress and severe dyspnea, a small amount of the fluid, sufficient to relieve the distressing symptoms, may be slowly withdrawn. Whenever possible it is advisable to have the effusion absorbed, as this seems to exert a favorable effect upon the general condition of the patient, although the exact way in which this autoserotherapy is exerted has not been definitely determined. It has been recommended that massive effusions be completely withdrawn, air or nitrogen gas being introduced at the same time, the gas injected replacing the fluid. In this way compression of the lung is maintained, and presumably there is less likelihood of the effusion becoming purulent. This replacement of fluid by gas impresses one as an unnecessary interference, subjecting the patient to the discomfort of frequent injections of gas which could be avoided, and at the same time producing conditions which favor the reaccumulation of the fluid. The encouragement of a gradual absorption of the fluid seems much more desirable, unless the process in the lung distinctly indicates the advisability of an artificial pneumothorax, regardless of the pleural effusion.

The favorable effects which frequently follow the absorption of effusions has led to efforts to bring about similar conditions artificially. The most common means of inducing autoserotherapy is by the use of large blisters (4" by 4" or 4" by 6"), care being taken to avoid breaking the skin in their application. The cantharides blisters are applied until distinct redness of the skin develops, the time required varying in different individuals from one to three hours, when the plaster is removed and the formation of the vesicle assisted by means of hot, moist compresses. After the blister is formed a carefully applied absorbent cotton dressing is necessary to prevent its rupture, and to allow the serum contents to be absorbed.

In the treatment of tuberculous effusions the injection into the pleural space of various substances has been recommended, with the object of curing any tuberculous disease of the pleura. Among the substances suggested are iodoform and glycerin (10 to 20 mls of glycerin containing a 20 per cent. suspension of iodoform), formalin and glycerin, iodine and glycerin, and similar mixtures and combinations. Care must be used to avoid injecting



substances which permit of ready absorption with the development of general toxic effects. The advisability of injecting anti-septic solutions into pleural effusions is still open to question, further experience with this method of treatment being necessary before it can be proved to be without danger, and to possess distinct advantages over the present methods of treatment. When the effusion becomes purulent it must be treated as any other form of empyema, regardless of the pulmonary process, but so long as it is serous it may be left in the pleural space indefinitely, the danger of permanent contraction of the lung or serious diminution of expansion being so remote a possibility that such complications may be disregarded. It is astonishing to find how quickly a lung which has been compressed for a considerable time will regain its normal or nearly normal expansion when the compression is removed, provided there has been no marked inflammatory thickening in the pleura or extensive disease in the lung.

**Tuberculous Pneumothorax.** This complication may occur in patients with only a moderate phthisical infection, but it is usually met with in advanced cases with cavity formation, the thin walls of which rupture with the consequent passage of air into the pleural space. As cavities usually contain infective material, which also gains entrance to the pleural space with the air, it is extremely rare to find a pneumothorax which does not become a pyo-pneumothorax within a very short space of time. In treating this condition this fact should always be borne in mind, as the pneumothorax alone calls for treatment in only a very small proportion of the cases.

*Treatment.* The occurrence of pneumothorax usually is accompanied by pain varying in degree from one so slight as to pass almost unnoticed to one of agonizing severity. In some cases, however, this symptom is entirely absent, and the presence of pneumothorax may be entirely unsuspected, being discovered only during the course of a routine examination, or it may be suspected only on account of the sudden development of dyspnea. The pain, if severe, is best treated by morphin, and it may become necessary to withdraw some of the air from the pleural space by means of an aspirating apparatus, if the dyspnea is very severe or if pain is unrelieved by morphin. The symptoms of shock, which occasionally accompany the onset of pneumothorax, may call for fairly active stimulation.

When considering the advisability of removing the air from the pleural space, or of aspirating a serous effusion which may develop secondarily, it must be remembered that one of the essentials of a cure of pneumothorax is the closure of the pulmonary opening. The closure of this opening is more likely to be secured if the lung remains compressed, whether secured by the air or by fluid in the pleural space. When the symptoms demand relief from the intrapleural pressure alone, a sufficient quantity of the air or serous fluid should be removed to relieve the urgent symptoms, and the evacuation never carried out to a point where re-expansion of the compressed lung will result.

The further treatment of the case will have to be determined by the extent of the lesion, the general condition of the patient, the amount of pus which develops in the pleural space, and the severity of the symptoms. Aspiration of the pus may be all that the condition of the patient will warrant. In those cases where it would seem advisable to prevent the re-expansion of the lung, air or nitrogen gas may be introduced simultaneously with the aspiration of the pus. By this method the removal of the pus may be secured, and at the same time the possibility of re-opening the pulmonary fistula can be avoided. As the outlook for recovery in these cases is very poor, it seems as if the best hope for recovery would depend upon the removal of the pus by the resection of the rib and the insertion of a drainage tube, when the patient's general condition permits a deliberate operation of this sort. The selection of the site of operation and the method of drainage should be left to the decision of the surgeon.

**Mixed Infections.** Many cases present a group of symptoms, such as marked elevation of temperature of a hectic type, night-sweats, chills, emaciation, general malaise, digestive disturbances, and purulent expectoration, which many clinicians believe are indicative of the presence of some infective organism other than, or in addition to, the tubercle bacillus. While in some instances this is undoubtedly true, more recent investigations would tend to show that this group of symptoms may be produced by the tubercle bacillus alone. In advanced pulmonary tuberculosis there are usually large areas in the lungs lined with breaking down caseous material or fibroid tissue, which offer a favorable nidus for bacterial growth; and while these secondary

micro-organisms may not gain access to the blood-stream, there is no reason why their soluble toxins may not enter the general circulation, and be responsible for the symptoms characteristic of this complication. It would seem wise to consider the foregoing group of symptoms as indicative of mixed infection, even if at times tuberculosis alone may possibly be responsible for their occurrence.

*Treatment.* These cases call for a line of treatment suggested in the sections on pyrexia and night-sweats, remembering that the low temperature in the mornings may call for treatment with warm drinks, hot applications, and extra coverings, just as the febrile period of the day calls for cold applications. These cases require abundant nourishment, and under no conditions should the amount of nutriment be diminished merely because the temperature is elevated.

The results from the use of bacterial vaccines are very striking in a few cases, but so far we have no means of determining which are the cases in which these favorable results may be expected. In the majority of instances no effect is obtained from vaccines, and in a few cases harm seems to result from their use. In the light of our present knowledge, if the usual means at our disposal for treating this condition prove unavailing, the bacterial vaccines may be tried cautiously, although one will be saved considerable disappointment in the majority of cases if not too much is expected from their use. Whenever possible it is advisable to employ auto-genous vaccines, although occasionally striking results may follow the use of stock mixtures of the bacteria. The method of procedure recommended is to prepare a bacterin containing all of the micro-organisms in a representative sample of sputum, the initial dose containing 25 million bacteria, which is increased by 20 per cent. every three to five days. Care should be taken to avoid producing a reaction, the best guide to the proper dosage being the evidence of improvement of the symptoms. Repeated examinations of the sputum should be made and the bacterin altered to correspond with the changes in the bacterial content of the expectoration.

**Nephritis.** Many cases of pulmonary tuberculosis in which the disease is of long standing present evidence of a mild grade of chronic parenchymatous nephritis. The renal disease

may even be the actual cause of death in a small proportion of cases. The disease of the kidneys may be entirely unsuspected, and discovered only in the routine examination of the urine. It is very infrequent to find the general symptoms and signs usually associated with disease of the kidneys, with the exception of anemia and gastro-intestinal disturbances. The diarrhea occasionally met with in advanced tuberculosis may have its origin in the decreased elimination by the kidneys.

In spite of the relative frequency of albumin and casts in the urine in advanced pulmonary tuberculosis, probably due to the prolonged elimination of toxins of various origins, the function of the kidneys seems to be fairly well maintained in a large proportion of the cases, if one may judge from the results obtained from the application of the functional renal tests.

In the advanced cases which show large quantities of albumin and a relatively small number of casts in the urine, one would naturally suspect amyloid changes in the kidneys, which are frequently accompanied by similar changes in the spleen and liver.

*Treatment.* The treatment of nephritis in the tuberculous will usually call for the exercise of judgment in restricting the amount of protein, and at the same time maintaining the general nutrition by increasing the amount of fats and carbohydrates. Many of the cases in which the renal process is not extensive, and the kidneys are still functioning fairly well, require very little in the way of treatment directed toward the kidneys other than that obtained by a change of diet. Where the general health appears to be affected by the renal disease or where there are symptoms definitely due to the renal disease, a more active course of treatment may be demanded. If the symptoms of nephritis are urgent, it may be necessary to ignore temporarily the tuberculous process, and to institute treatment regardless of its presence. Some care must be used in employing hot baths in the cases in which they may be indicated, when any marked breaking down in the lungs exists. Severe hemoptysis may result from the injudicious employment of hot baths or packs in such cases.

Where increased elimination by the intestines is desired, the physician in charge will have to decide in the individual



case the point to which the active catharsis may be carried without seriously endangering the patient by the depletion and the attendant loss of nutrition and strength which it so frequently produces. Care must also be used in these cases to make sure that the open-air treatment is not accompanied by chilling, and to see that extra precautions are used to make sure that the patient has sufficient bedding, and even artificial heat, if necessary, when in the open air.

**Intestinal Tuberculosis.** Approximately 70 per cent. of all far advanced cases of pulmonary tuberculosis have been found to have tuberculous ulceration of the intestines when examined *post-mortem*, some reports showing as high as 98 per cent. This gives an indication of the frequency of this complication in the advanced or terminal stages of the disease. It is impossible to state how frequently it may be present in the early stages, or even in the advanced cases, as we have no means of determining how long this condition may have existed previous to death. Unfortunately we have no reliable method of diagnosing the condition, for while diarrhea, pain, tenderness, and rigidity, especially in the right iliac fossa, may be present in tuberculous ulceration of the intestines, it may occur without any of the above symptoms, or the symptoms may occur without any ulceration. The presence of tubercle bacilli is valueless, if there is tuberculosis in any other part of the body, and the presence of occult blood in the stools is of no practical value. Hemorrhages from the bowels have been considered by some writers as an absolute indication of tuberculous ulceration, but the diagnoses in the cases reported were merely assumed, as there was no direct examination of the intestines to support such an opinion. When the patient has been progressing favorably, or has remained stationary for a considerable period of time, and suddenly either ceases to improve or becomes progressively worse, without any definite patent cause, and without any apparent change in the pulmonary process, the possibility of tuberculous ulceration should always be suspected.

**Treatment.** The treatment of this condition is extremely unsatisfactory. The main indication is the removal from the diet of all foods which leave much residue, especially those in which the non-nutrient portion is of an irritating character.



An absolute egg-and-milk diet probably meets the indications better than any other, peptonizing of the milk aiding materially in securing the desired conditions. A bland soothing oil seems to give considerable aid in some cases, and even castor oil combined with tincture of opium is of considerable value in some cases, especially when diarrhea accompanies the condition.

In the majority of cases opiates in some form will be found an essential part of the treatment. The intestinal antiseptics are of very little value in the treatment of these ulcerative processes, although they may be tried in the patients in which there are certain symptoms indicative of intestinal tuberculosis, but in whom it is impossible to make a positive diagnosis. Creosote at times will relieve many of the intestinal symptoms associated with gaseous fermentation. Bismuth subnitrate, subgallate, or salicylate will help considerably in the cases associated with diarrhea, the subnitrate being the most satisfactory salt.

The ulcerative type is the most common of the tuberculous processes in the intestinal tract, but occasionally the hypertrophic form is encountered, with symptoms usually indicative of stenosis or incomplete obstruction of the intestine. In this type medicinal treatment is valueless.

Surgical intervention should always be considered in any case in which the diagnosis can be made with relative certainty, and in which the general condition of the patient warrants such a procedure, or where the condition has led to repeated hemorrhages or perforation. The operative treatment is naturally more clearly indicated in those cases in which the intestinal lesion is primary, or where the signs indicate a very slight or quiescent pulmonary lesion.

**Tuberculous Laryngitis.** In a work of this kind it is not the place for a detailed description of the various methods of treating tuberculous laryngitis, and yet it might not be amiss to offer a few suggestions in regard to the treatment in general. The majority of patients with tuberculous laryngitis only obtain an improvement of the laryngeal symptoms when the general health is built up—in other words, the laryngeal condition is best treated by measures directed toward the general welfare of the patient, rather than by local measures alone. Most cases

do better without much treatment of the larynx itself, unless there are very definite indications for such intervention. Absolute vocal rest, counter-irritation in the form of small blisters to the throat, a bland protective oil spray for the larynx, and the avoidance of unnecessary local applications, seem to be the methods which promise the best results in the average case. As stated above, the improvement of the patient's general health, with its consequent increased resistance to the disease, secured by sufficient fresh air, food and rest, is, after all, the most essential factor in the treatment of laryngeal tuberculosis.

**Fistula in Ano.** The development of *fistula in ano* is of considerable diagnostic importance, and the presence of this condition should at once direct attention to the probable existence of pulmonary tuberculosis.

When this condition is present in a patient suffering from pulmonary tuberculosis, the value of surgical interference is very questionable. It is not an uncommon experience to find an increased activity of the pulmonary process following, and apparently caused by, operative measures applied for the purpose of closing these fistulæ.

The presence of a small fistula is not a serious detriment to the health or comfort of the patient, and it will be found that they not infrequently heal under simple local cleanliness and the general hygienic measures adopted for the arrest of the pulmonary process.

It has been frequently noted that many cases of pulmonary tuberculosis appear to do very much better, so far as their general health and pulmonary process are concerned, as long as the fistula continues discharging. When the fistula becomes closed, without healing from the bottom up, an exacerbation of the pulmonary disease frequently results.

#### ASSOCIATED DISEASES.

**Cardiac Disease.** For many years the opinion was held that an antagonism existed between valvular heart disease and pulmonary tuberculosis, this belief being founded upon observations made by numerous early writers, who held that phthisis rarely occurred in subjects of valvular heart disease, or that if it did occur the pulmonary process followed a be-

nign course. More recently repeated observations have conclusively shown that valvular cardiac disease exerts very little, if any, influence upon tuberculosis of the lungs, either as an inhibitive or as a curative influence, even if there is no loss of compensation. In anyone suffering from valvular disease of the heart, one must be extremely cautious in making a diagnosis of pulmonary tuberculosis solely upon the physical signs in the chest. It is not at all uncommon to find localized areas of congestion, even over the upper portions of the left lung, which suggest the presence of a tuberculous focus. When the patient presents, in addition, the history of prolonged cough, dyspnea, and hemoptysis, the presence of pulmonary tuberculosis is naturally suspected. Where there is loss of compensation, dilatation of the heart, or hydrothorax the diagnosis of pulmonary tuberculosis should only be made in the presence of irrefutable evidence, unless the pulmonary signs persist after compensation has been restored, or the condition of the heart and pleura more nearly approach the normal.

The treatment of cardiac disease when associated with pulmonary tuberculosis does not differ in any way from that of the uncomplicated cases. The use of digitalis is not contra-indicated, but drugs of this type should be administered with caution in the cases in which there is a history of hemoptysis. In certain persons suffering from hemoptysis, in which there is evidence of cardiac weakness, the employment of cardiac stimulants not infrequently is followed by relief from this symptom.

**Syphilis.** A double infection with tuberculosis and syphilis is not at all uncommon, even active tuberculous disease being found in individuals presenting evidence of lues, either of the secondary or tertiary stages. When either the clinical manifestations or the Wassermann test calls for active anti-syphilitic treatment, this should be given regardless of the pulmonary process. Active treatment is especially indicated when there is distinct clinical evidence of the luetic disease.

The presence of pulmonary tuberculosis is not a contraindication to intravenous medication, but it is necessary to inject the solution more slowly, and at the same time to make frequent examinations of the blood-pressure, in order to make

sure that it does not become unduly elevated. Several observers have shown that the intravenous injections of salvarsan are not accompanied by a rise of blood-pressure, but one should avoid any possibility of danger from this source.

There is a widespread impression that iodides are dangerous drugs to employ in pulmonary tuberculosis, for fear of causing a breaking down of the connective tissue barrier which may have been established about the tuberculous focus. The grounds upon which this is based appear to be more theoretical than real, but even if this danger does exist when the patient is suffering from marked tertiary lesions or any serious complication calling for the administration of iodides, the pulmonary process should be ignored, and the risk taken, if such exists, of causing a breaking down in order to relieve the patient from the added aggravation of the secondary disease.

**Diabetes.** The view has been held for many years that diabetes mellitus predisposes to the development of pulmonary tuberculosis, and that this disease is the cause of death in a large proportion of diabetics. The proof submitted is, however, by no means conclusive that tuberculosis occurs more frequently in diabetics than in the general population at the same age periods.

The treatment of diabetes has been so hopeless in the past that the appearance of glycosuria in a person suffering from tuberculosis was always considered of a very grave significance. With the advent of the Allen treatment, which promises so much in diabetes, the question arises as to whether the treatment is applicable in pulmonary tuberculosis—whether a treatment based upon starvation is applicable to a class of cases in which hypernutrition is the most important part of the treatment. Recent investigations show that the Allen treatment may be applied in the tuberculous with decided benefit, if carried out with care and judgment, and with the patient under careful observation and complete control; in other words, under the only conditions under which the Allen treatment should ever be employed. As in nearly every other associated grave disease, it is the tuberculosis which must be ignored when the point is reached where one must decide as to which process requires the more



energetic treatment. The general improvement which follows relief from the glycosuria more than repays the slight risk which one runs by the few days starvation, or the short period of limited food.

**Pregnancy.** Many tuberculous women apparently suffer no harm from, and even appear benefited by, pregnancy, but practically every one is unfavorably affected by parturition, inasmuch as childbirth frequently leads to an exacerbation of the tuberculous disease, even when it has been quiescent for a considerable period of time. In view of the serious results which may follow child-bearing, when consulted by a tuberculous woman as to the advisability of marrying, she should be warned of the dangers attending this function, unless the disease apparently has been cured for several years, and the greatest care is used during pregnancy and parturition. Many eminent authorities contend that the married tuberculous woman should not only be warned of the dangers attending childbirth, but should be instructed in the measures for preventing conception.

The majority of cases which call for advice are tuberculous women who are already pregnant, and who desire to know whether any additional precautions are necessary to safeguard their health. Many physicians insist upon emptying the uterus whenever there is evidence of activity in the pulmonary process. The question of whether pregnancy should be interrupted must be decided in the individual case, depending upon the general conditions under which the patient lives, whether she is able to receive the care and treatment necessary and has sufficient intelligence to carry out the necessary measures, whether there are other children, whether the disease is markedly active, and the duration of the pregnancy.

During pregnancy a tuberculous woman should be kept under very careful observation, in order that her general nutrition be maintained at the highest possible level, and that the first indication of activity may be detected. Many writers believe that upon the first appearance of any unfavorable symptom suggestive of active tuberculous disease, when it occurs prior to the fifth month of pregnancy, the uterus should be emptied. After the fifth month the case should be treated



expectantly, and labor be made as easy as possible, even inducing premature labor two weeks before term in some cases.

Many women go through pregnancy without any unfavorable symptom referable to the pulmonary disease, only to have it become violently active during or shortly following the puerperium. Every woman who has suffered from tuberculous disease, no matter how long it has remained quiescent, should be treated as if an active case of tuberculous disease existed, for a period of one to two months following parturition. The development of activity in the pulmonary process may be so insidious as to evade detection for a considerable period, even when under careful observation. The enforced period of absolute rest eliminates this danger as far as it is possible by our present methods of treatment.

Tuberculous women should not nurse their children, notwithstanding the disadvantages of artificial feeding, except in very exceptional cases, and never when the mother's sputum is known to contain tubercle bacilli. The extreme susceptibility of infants to bacterial infections is almost certain to lead to an implantation from the intimate association with the mother, particularly in breast-fed infants, even if the danger of a milk-borne infection could be excluded. It has been shown that milk from a tuberculous mother occasionally contains tubercle bacilli, and this fact constitutes an additional risk to the child.

### CHRONIC NON-TUBERCULOUS PULMONARY INFECTIONS.

The chronic non-tuberculous pulmonary infections have been receiving more attention in recent years than was formerly accorded them, and while evidence is accumulating to indicate that such conditions actually exist, it would be premature at this time to describe definitely their frequency, pathology, symptomatology, diagnosis, or treatment. The reported cases seem to indicate that the general symptomatology closely resembles that of tuberculosis, for which they are usually mistaken, the disease being accompanied by chronic cough, expectoration, slight elevation of temperature, and gradual loss of weight and strength. Night-sweats and

hemoptysis have been reported as occurring during the progress of the disease. Non-tuberculous infections of the type under discussion usually follow some acute infection, especially one in which there has been some affection of the respiratory tract, or they may result from lobar or bronchopneumonia, more commonly following repeated attacks of pneumonia. It is not uncommon to find a chronic cough and expectoration in children who have been the subject of some acute infectious disease, such as measles, pertussis, or scarlatina, or have passed through an attack of pneumonia. These children are usually underweight, anemic and weak. On examination they are found to have an elevated temperature, not confined to the afternoon but seemingly fairly constant, the pulse is rapid, and on examination of the chest localized râles are revealed, confined to one or the other lower lobes. The râles may become apparent only after cough, or as the result of change of position, especially when the patient is examined in the inverted position. A moderate degree of limitation of pulmonary excursion may be present, with impairment on percussion, diminished or suppressed breath-sounds, and slight increase or no change in the voice-sounds over the affected area. These signs may persist for months or even for years with recurrent exacerbations of the original symptoms. In adults a similar condition may be found, in which one usually obtains a history of cough and expectoration, frequently dating back to childhood, the condition being ascribed to an attack of pneumonia or to one of the acute infectious diseases.

The pathology of the process has not been definitely determined, but from the clinical study it probably consists of an interstitial thickening, the result of hyperemia and cellular exudation. Until more is known of the pathologic factors at work, a doubt must remain as to whether these cases should not be considered as early or slight cases of bronchiectasis. For the present it would seem more rational to consider them as cases of a definite pathologic process, until further evidence has been collected to establish the existence of a probable relationship to any other disease.

The *treatment* apparently followed by the best results is that suggested for bronchiectasis, namely postural changes,

to facilitate the expulsion of the accumulated expectoration, and the upbuilding of the patient's general health and strength. Further study may confirm the finding that many of these cases are due to specific bacterial infection (influenza bacillus or streptococcus, for example) in which event beneficial results possibly may be obtained from the use of bacterial vaccines.

### PULMONARY CONGESTION.

Active congestion of the lungs may perhaps occasionally occur as a primary disease, but, as a rule, it is secondary to some other process, constituting the first stage of pneumonia, accompanying to a greater or less extent all acute inflammatory and many tuberculous processes in the lungs, pleurisy with effusion; it may also occur in consequence of the too rapid withdrawal of pleural effusions, or from the inhalation of irritating gases or fumes. A primary form has been described which has been recognized as a distinct disease process (Woillez's disease), which recent investigations indicate is probably merely an abortive type of pneumonia.

The hyperemic areas of the lung are dark-red in color, of somewhat increased resistance, and diminished elasticity, although the pulmonary tissue is still air-bearing. The cut surface exudes an increased amount of bloody fluid, usually dark-red in color. Microscopically, the alveoli are seen to contain serous exudate, leucocytes and desquamated epithelial cells, the capillaries being engorged with blood.

The passive form results from some obstruction to the flow of blood from the lungs to the left side of the heart, as a result of cardiac disease, such as mitral disease, or insufficiency of the left ventricle, leading to a damming back of the blood in the pulmonary veins or where some mechanical obstruction to the flow of blood through the pulmonary veins exists, such as pressure from some mediastinal growth, or thrombosis of the veins. Long standing cardiac disease may result in a chronic passive congestion of the lungs of an extreme grade, with the production of certain changes in the lung, described under the name of brown induration. The lungs are large, firm, inelastic, rather fragile, and of a russet-

brown color, due to the deposit of blood-pigment. The blood-vessels are usually engorged, and small parenchymatous hemorrhages may be present. Microscopically, the capillaries are dilated, and there are possibly small areas of ecchymosis and a moderate increase of the interstitial tissue containing pigment granules; these are seen also within the alveoli, either as free particles or included in epithelial cells or leucocytes.

The *symptoms* resulting from pulmonary congestion are cough, dyspnea, and cyanosis. Cough is a very frequent symptom and may be unaccompanied by expectoration, or with the production of a slight mucoid material. The sputum frequently contains blood, in the form of slight bloody streaks, bloody and frothy mucus, or as actual frank hemorrhages. The pigmented epithelial cells ("Herzfehlerzellen") are frequently present in the sputum. The cough and expectoration may be aggravated or modified by the development of a secondary bronchitis. The dyspnea is usually an early and persistent symptom, and it may become extremely severe. The reclining position usually intensifies the dyspnea when due to passive congestion, the relative relief in the upright position being quite a striking feature. Cyanosis varies greatly in these cases with the degree of pulmonary stasis, but it is almost always present to some extent.

Passive congestion of itself may give rise to no *physical signs* which can be elicited on examination of the chest. The presence of fluid in the air-passages, which usually accompanies the process, gives rise to râles, as a rule most marked over the lower portions of the lungs. The hypostatic type of passive congestion results from loss of tone of the pulmonary vessels, failing circulation, and the effects of gravity; this type of congestion is met with in chronic debilitating diseases, poisoning, coma, old age, etc. In these cases the dependent portions of the lung are the seat of a congestion which is more marked than in the other portions. Hypostatic pneumonia may develop in these areas of congestion as the result of bacterial invasion.

When the pulmonary congestion develops suddenly from rapid loss of cardiac power, the symptoms usually are very severe, and the hard, dry cough, intense dyspnea, orthopnea,

cyanosis, and oppressive feelings in the chest have led to the misnomer of "cardiac asthma" being applied to this condition.

### TREATMENT.

The treatment of this process depends upon the nature of the underlying condition with which it is associated. Where cardiac disease or failing circulation are responsible, diffusible stimulants are often of considerable help. Much may be done to prevent the development of congestion of the hypostatic type by frequent change of position in those in whom it is likely to occur, or by propping up in the erect position. In the treatment of the aged for any condition which may confine them to bed, and in prolonged wasting diseases, this precautionary measure should be constantly kept in mind. When the pulmonary engorgement is of a severe type, and complicated by dilatation of the right heart, the symptoms may become so urgent that venesection will be necessary, in order to avert cardiac failure.

In the ordinary type, dry cups to the chest, counter-irritation with mustard or turpentine, diffusible heart stimulants, expectorants, diuretics, and purgatives will usually meet the requirements. When associated with or due to some other process, the treatment will have to be modified to meet the existing conditions.

### PULMONARY EDEMA.

The transudation of a serous fluid into the alveoli, bronchioles, and interstitial tissues of the lung may occur secondarily to congestion, or as a terminal process in death from any cause. It is rare to find congestion of the lungs without a certain amount of edema, irrespective of the cause of the congestion.

There is a primary type (acute suffocative pulmonary edema) which develops in an acute manner, usually in subjects of arteriosclerosis, chronic disease of the heart or kidney, during the course of acute infectious diseases, or rarely as an idiopathic process. The rapid removal of large collections of fluid from the pleural space or the peritoneal cavity also has in rare instances been followed by acute pulmonary edema.



Pulmonary edema seems to depend upon an increased pressure within the pulmonary capillaries, combined with an increased permeability of the vascular walls of some undetermined nature. The appearance of the lungs varies with the degree of pulmonary congestion with which it is so very frequently associated. In addition to congestion, the lungs show the presence of an excessive amount of fluid, which exudes freely from the cut surface, and, microscopically, the alveoli and interstitial tissues contain an excessive amount of serous fluid, in addition to such changes as have been described under congestion.

Edema of the lungs is manifested clinically by the more or less profuse expectoration of a frothy serous fluid, rich in albumin, usually blood-tinged, from the associated congestion. The dyspnea may be severe, the cough very distressing, and cyanosis of varying degrees of intensity is frequently present. Upon examination, fine moist râles are audible over both lungs, or, if the amount of serous exudate is excessive, coarse, bubbling râles may replace or obscure the finer râles, in either instance the signs of moisture in the lungs being most marked at the bases. The breath-sounds are usually suppressed or obscured by the râles, and on percussion there may be slight impairment of resonance at the bases, depending upon the severity of the process.

In acute pulmonary edema the attacks usually are very severe, coming on suddenly, with very slight or no premonitory signs, the patient being suddenly seized with intense dyspnea, labored breathing, and orthopnea. The cold extremities, free sweating, and cyanosis indicate the grave nature of the attack. The sense of oppression in the chest, or suffocation, also is usually present. The cough is extremely distressing, and accompanied by the expectoration of a frothy, thin, watery material, pinkish in color; ordinarily this fluid is moderate in amount, but it may be so excessive as to gush from the mouth, the quantity expectorated reaching even as high as 1 or 2 quarts (1 to 2 l.) during the attack. Death may result from the first attack, although recovery from the initial edema is more common, to be followed by repeated attacks until death intervenes, either during the attack or from some associated condition between attacks.

### TREATMENT.

The treatment of edema of the lungs depends upon the nature of the underlying cause, as described under Congestion. In the acute suffocative types, due to cardio-renal, or cardio-vascular disease, an effort must be made to re-establish the balance between the working power of the two sides of the heart, the loss of which is a most important if not the only factor involved in the development of the pulmonary edema. The lost strength of the left ventricle is the essential feature of the process, although the underlying cause may be disease of the coronary arteries, arterial hypertension, or a combination of both. To restore the circulatory equilibrium is a far from easy matter, requiring a careful study of the nature of the pathologic process responsible for the disproportion between the action of the right and left sides of the heart.

Before resorting to the use of cardiac stimulants it is advisable first to try the effect of such general measures as rest and quiet, counter-irritation, dry cups to the chest, hot packs to the extremities, and increased elimination by the kidneys, skin, and intestinal tract. There is some question of the advisability of using morphine in these acute cases, but it may be employed in combination with atropine, if care is exercised. When cyanosis is extreme, oxygen inhalations may be found useful. Cardiac stimulants require very careful handling in this condition, to avoid doing more harm than good. When arterial hypertension is apparently responsible for the left ventricular failure, relief may be obtained by the employment of strophanthin and nitroglycerin or amyl nitrite. In the event of failure from the measures suggested, it may become necessary to resort to digitalis, camphor, ether, ammonia, and caffeine, or if signs of enlargement of the right heart develop, with marked cyanosis, venesection may be necessary to relieve the immediate dangers of a fatal termination.

### PULMONARY ABSCESS.

Abscess of the lung may arise from disease of the lungs, bronchi, or pleura, from the extension of a suppurative process in neighboring organs, or by metastasis from a septic process

in some remote portion of the body. The disease which seems to be most frequently responsible for their production is lobar pneumonia; bronchopneumonia appears to be a rather uncommon cause when considered in its primary forms. When bronchopneumonia results from the aspiration of foreign bodies, or arises from bronchiectasis, etherization, or putrid bronchitis, it probably plays a prominent rôle in the formation of the abscesses resulting from such conditions. The abscesses arising from localized suppurative processes in the pleura probably occur more frequently than is generally supposed, although, in the majority of cases, it is extremely difficult to say whether the pleural collection of pus has extended into the lung, or the pulmonary abscess has extended to the pleura. Necrosis of the vertebræ, suppuration of the bronchial glands, or septic processes in the upper abdominal cavity may extend directly to the lung, with the formation of abscesses. Septic thrombi from processes in other parts of the body may be carried by the blood-stream to the lung, where abscesses may arise at the point where the emboli find lodgment, from a breaking down of the septic infarct. The abscesses which may arise in the course of pulmonary tuberculosis have been considered under another heading (see p. 372), also those due to streptothricosis, actinomycosis, syphilis, and aspergillosis.

Abscess may occur in any part of the lung, although the lower portions are more frequently affected than the upper, usually being situated near the pleura (except those due to foreign bodies, bronchiectasis, etc.). The amount of inflammatory exudate or induration in the surrounding lung tissue depends upon the length of time the abscess has been present; in the earlier cases the pneumonia-like process is more extensive and is not sharply defined, gradually shading off into normal lung tissue. When the abscess is of long standing, this surrounding zone usually undergoes fibroid change, with the formation of a relatively narrow, dense, scar-like wall about the abscess cavity.

The pulmonary abscesses have the appearance of grayish, yellowish, greenish, or reddish-brown areas, which contain varying amounts of pus. The cavities vary greatly in size, from minute microscopic spaces to the dimensions of an entire

lobe. They may occur singly or as multiple abscesses, either in close proximity or widely separated. In recently formed or rapidly developing cavities the walls are usually rough, irregular, and necrotic, and not sharply defined from the surrounding lung tissue, which is almost universally inflamed. In older processes the surface of the cavity usually is gray, whitish, brown, or even black, and usually smooth, with slight irregularities, or occasionally lined with trabeculæ. The wall is dense and firm from the formation of connective tissue, and of varying thickness. The zone of inflammation surrounding the abscess also may undergo organization with the formation of a dense, firm, airless tissue. The older cavities may not contain pus, for the contents may be evacuated through the bronchi. When present the pus may be odorless, although usually it has a sweetish, sour, or foul odor.

Microscopic examinations may reveal exceedingly minute areas in which pus cells replace the normal alveolar tissue of the lung. These areas may undergo a healing process, with the complete restoration of function, or develop into well-marked abscess cavities. These microscopic abscesses are not uncommon in both lobar and bronchopneumonia. When there is distinct abscess formation, the walls are infiltrated with pus cells, which extend into the surrounding lung tissue. The contents of the lung cavity consist of detritus, pus cells, bacteria, shreds of broken down lung tissue, and elastic fibers. In the older abscesses the walls of the cavity and the surrounding tissue show evidence of new connective tissue formation, in the interalveolar, interlobar, peribronchial, and perivascular tissue. This formation of fibrous tissue may be so dense as to cause complete obliteration of the alveoli in the immediate neighborhood of the abscess.

The presence of an abscess should be suspected in any case of lobar pneumonia in which the elevation of temperature persists beyond the ordinary time for it to decline, and in which the expectoration becomes purulent, or the area of consolidation fails to resolve. At this time no definite physical signs may be present, the general condition of the patient and the irregular temperature suggesting empyema, caseous pneumonia, or abscess. The signs of a cavity may appear after the patient has suddenly expectorated a large quantity of

purulent material. This tendency to expectorate suddenly a large amount of purulent material at varying intervals is probably the most suggestive symptom of abscess of the lung, the evacuation of its contents being induced by change of position, coughing, laughing, or sneezing as in bronchiectasis. The symptoms of pulmonary abscess may present no special characteristic to suggest the presence of such a process. This is particularly true of those abscesses developing in the course of inflammatory diseases of the lungs, or as the result of an extension of a suppurative process to the lung.

The purulent material is, as a rule, not particularly offensive unless a certain amount of gangrene is present, but this rule is not absolute, as the pus may be extremely foul. The quantity varies greatly, is usually of a greenish color, and on standing has a tendency to separate into three layers, with a heavy layer of pus at the bottom, a zone of foamy mucus on top, and a fairly clear or cloudy yellowish stratum between. This tendency of the pus to form three layers, while very suggestive, is not absolutely characteristic of abscess, as it may also occur in material from a bronchiectatic cavity, or in an empyema which has been evacuated through a bronchus.

The finding of elastic tissue in the pus, especially when it shows a reticulated formation or alveolar arrangement, is indicative of a breaking down of the lung tissue, strongly suggestive of abscess. Care must be exercised in examining the sputum for elastic fibers to exclude food particles, as they constitute a not uncommon source of error. The elastic tissue may be visible to the naked eye, when the sputum is spread in a thin layer over a black background, as small yellowish masses. A better method for studying this tissue is by diluting the sputum with twenty volumes of water, to which a few drops of a saturated solution of potassium hydroxide have been added. The mixture is warmed until the sputum is dissolved, and the sediment obtained by centrifugation is mounted upon a slide and stained with Weigert's elastic tissue stain. By this method the elastic tissue fibers are stained a very dark blue or black, which permits accurate study of their alveolar arrangements, and avoids any possibility of confusing them with other constituents of the sputum, which occasionally may cause some confusion.



The *physical signs* vary very much, and usually are those indicative of dense, inflammatory exudate rather than cavity, especially when the condition is fairly recent. The signs of cavity may be elicited if the chest is examined shortly after the expectoration of a large quantity of pus. The abscesses which are overlooked are those in which evidences of cavity have been lacking, and on that ground the possibility of cavity ignored. It may be stated that definite signs of cavity with tympany, cavernous or amphoric breathing, bubbling metallic râles, and whispering pectoriloquy over a recent abscess are the exception rather than the rule.

A localized collection of râles, dullness on percussion, and slight bronchovesicular breathing may suggest the presence of abscess, such signs being due to the consolidation or cellular infiltration of the pulmonary tissue in the immediate neighborhood of the abscess. When the abscess is large and filled with fluid there may be absent or diminished breath- and voice-sounds, in addition to the dullness, over the area affected. With the evacuation of the pus, the signs of cavity may appear, only to disappear as the fluid reaccumulates.

The study of the case by means of stereoscopic skiagrams may be of considerable service in locating the abscess cavity, especially when it is of long standing. In recent abscesses the shadow corresponds to the physical signs indicating an area of infiltration rather than a cavity, on account of the dense zone of infiltration with which the cavity usually is surrounded. This method of study has the additional advantage of revealing the presence of foreign bodies, which may be responsible for the abscess formation, and plates taken immediately after the expectoration of large quantities of pus may reveal the presence of the abscess cavity. Abscesses of long standing may be indicated upon the plates as more or less irregularly rounded clear spaces surrounded by a definite dark shadow due to the surrounding dense connective tissue.

While complete recovery from undoubted pulmonary abscesses have been recorded, this is not the rule, the patient more commonly continuing to cough and expectorate pus even after the acute, severe symptoms subside, the fibroid changes induced in the surrounding lung tissue usually preventing a complete return to normal.

## TREATMENT.

The treatment of the acute condition consists in absolute rest in bed, fresh air and nourishing food (see Tuberculosis, p. 409). Care must be exercised to prevent the swallowing of the purulent expectoration, and the patient warned of the necessity of not allowing any of the expectoration to gain entrance to the esophagus. A mouth-wash should be provided, to keep the mouth and throat thoroughly cleansed. In some cases the patient unconsciously assumes the position which favors drainage, while others, in order to prevent cough and expectoration, habitually lie in that position which helps to retain the purulent contents of the abscess. Various positions in bed should be tried, to determine the posture most likely to favor the evacuation of the pus. When this has been determined the patient should be instructed to assume such a position at frequent intervals during the day, so that the accumulation of large quantities of pus may be avoided.

Various medicinal agents may be employed which favor the expectoration of the purulent material, such as ammonium chlorid, creosote, oil of eucalyptus, and oil of turpentine, especially if the sputum is very offensive. Steam inhalations may also give considerable relief, especially when combined with various medicinal agents (see Bronchiectasis, Acute and Chronic Bronchitis, pp. 330-344). The derivatives of opium should be absolutely avoided, except when there is a constant, dry, unproductive cough, in which event it may be necessary to give morphin  $\frac{1}{8}$  grain (0.008 Gm.), heroin  $\frac{1}{12}$  grain (0.0054 Gm.), or codein  $\frac{1}{4}$  grain (0.016 Gm.), the doses being repeated, if necessary, to secure sufficient sleep.

In the acute cases, when the patient does not show signs of marked sepsis, when the pus is not foul, and when the physical signs and sputum examinations indicate very little breaking down of the lung tissue, the medical treatment suggested may be tried for several weeks, in the hope of securing a spontaneous cure.

When the pus is foul, and there is evidence of an active destructive process in the lung, the advisability of surgical interference must be considered.

Surgical treatment is not indicated, as a rule, in abscess cavities of long standing, and in multiple, widely-separated

abscesses, although the question is one to be decided in the individual case. Recently very favorable results have been reported from the use of artificial pneumothorax in the treatment of pulmonary abscesses, instead of the open operation, with direct incision and drainage of the abscess cavity. The amount of induration about the abscess and the duration and location of the process must be taken into consideration in deciding between these two methods of treatment.

In cavities of very long standing, attended by marked symptoms of an unfavorable character, it may be necessary to consider the advisability of extensive resection of the ribs, or even pulmonary resection.

The results of operative interference in cases of pulmonary abscess are more satisfactory than formerly, owing to improved technic and the newer methods for the prevention of pulmonary collapse when the chest wall is opened. The use of stereoscopic skiagraphs has also proven of assistance in the surgical treatment of these cases by permitting a more exact localization of the abscess in obscure cases than was formerly possible.

### PULMONARY GANGRENE.

What has been said in regard to the etiology of abscess applies equally to gangrene, except that the latter is less frequent as a result of lobar pneumonia, and is more likely to be due to the aspiration of putrefying material, such as may occur during operations upon the nose, mouth, or throat, while the patient is under ether, or as a result of diphtheria, cancer of the tongue or jaw, or new growths of the esophagus. While numerous bacteria have been described as the cause of gangrene, this etiologic relationship has never been definitely proven. Gangrene of the lung is consequent to serious interference with the blood-supply of a portion of the lung, such as thrombosis caused by bacterial infection or embolism, or by infection with micro-organisms of a peculiarly destructive or putrefactive type. While an entire lobe or greater portion of a lobe may share in the gangrenous process, the small localized form is more common, varying in size from a pea to an orange. They vary in color from a greenish gray to greenish brown or black, the tissue being very fragile, pulpy,

and may contain cavities. The odor is extremely foul. The cavities usually have rough, shreddy walls, and not infrequently contain fluid of a greenish or brownish color, with a very offensive odor. The lumen of the cavities may be traversed, or the walls lined, with bronchi or blood-vessels. The gangrenous areas are surrounded by a zone of hyperemia or consolidation, beyond which the pulmonary tissue is edematous. Occasionally the process may be walled off from the surrounding tissue with the formation of a cavity, but in the majority of cases the process continues to spread. When studied under the microscope the alveoli are found to contain desquamated epithelial cells, serum, numerous erythrocytes, a few leucocytes, and a small amount of fibrin. The formation of connective tissue may be present in the surrounding lung, similar to the process accompanying pulmonary abscess. What has been said of pulmonary abscess in regards to the location and number of the lesions, and the changes in the surrounding lung tissue and pleura, applies equally to gangrene. The clinical symptoms of the two processes are almost identical, except that in gangrene the depressing effect upon the patient's general health is much more marked, and, as a rule, the prostration being more severe. The sputum usually is more offensive than in abscess, and more likely to be of a dark, chocolate-brown color, due to the admixture with blood. At one time the presence of elastic fibers in the sputum was considered as indicative of abscess rather than gangrene, but the results of further study have shown this assumption to be unwarranted. Small masses or shreds of the lung parenchyma are more commonly present in the material expectorated in gangrene than in abscess of the lung.

The clinical differentiation between abscess and gangrene rests upon the odor of the expectorated material in the majority of instances, which gives one some conception of how closely the two conditions resemble one another, and how inadequate are our present means of differentiation.

The *treatment* of gangrene differs in no way from that of abscess, except that as the process is of a more acute and malignant character than many abscesses, one is not justified in adopting any plan of expectant treatment for any length of time. The diagnosis of gangrene calls for active and radical



treatment, being influenced to a certain degree by the amount of lung tissue involved and the etiologic factor responsible for the condition in the individual case.

### PNEUMOCONIOSIS.

The inhalation of various metallic and mineral dusts, which are either expectorated or deposited in the tissues of the lungs, may give rise to very annoying symptoms, due to mechanical irritation by the minute particles, but the most serious result of such inhalations is the irritation and inflammation induced thereby, which make the subject susceptible to other infections of the lungs. The various dusts which may give rise to pulmonary symptoms cannot be considered in detail in a work of this kind, as such a study would include an exhaustive review of the working conditions in nearly every trade. The dusts usually responsible for pneumoconiosis may be conveniently grouped under the headings of coal (anthracosis), metallic (siderosis), and stone (chalicosis and silicosis). The lungs of all inhabitants of large cities and those living near manufacturing plants burning coal as a fuel show pigmentation of the pulmonary tissue with carbon-particles to a greater or less extent. Carbon deposits in the lungs of sufficient extent or degree to warrant the name of anthracosis are found only among coal-miners, coal-heavers, and men in similar occupations. The metallic dusts are responsible for pneumoconiosis in workers whose occupation exposes them to the inhalation of finely divided particles of steel, iron, tin, and similar metals, commonly consequent to the trades of grinding, filing, and polishing. The trades of stone-cutting, stone-crushing, blasting, and pottery work are productive of the large majority of pneumoconiosis due to particles of stone. The inhalation of vegetable or animal fibers, such as hair, wool, flax, cotton, flour, cereals, and sawdust, while producing very persistent irritation of the air-passages, is not so likely to induce secondary infections.

The foreign particles may be deposited in the lungs in enormous quantities, without causing any symptoms or changes in the lungs other than those due to their presence, such as color changes and grittiness.



When changes in the lungs do occur they are not in any way peculiar or characteristic, for they include bronchitis, chronic induration, fibrosis, emphysema, bronchiectasis, abscess, adhesive pleurisy, and enlarged bronchial glands, either singly or in various combinations. The principal danger from dusty occupations apparently does not lie in the invasion of the lung by dust particles themselves, but in the infection of the lung with various bacteria, favored by the irritation resulting from the dust. Dusty occupations predispose to diseases of the bronchopulmonary system, as has been repeatedly shown by morbidity and mortality statistics. Among workers in dusty trades and occupations it has been repeatedly shown that the deaths from pulmonary tuberculosis are greatly in excess of the average, with the exception of those working in coal-dust. This relative infrequency of pulmonary tuberculosis among both soft and hard coal miners has never been satisfactorily explained, although the truth of the observation has been repeatedly confirmed.

The clinical picture depends entirely upon the nature of the changes set up in the lung as a result of the inhalation of the dust, the symptoms being the same as in bronchitis, bronchiectasis, and so forth, otherwise acquired, in which dust is not in any way concerned in the etiology. The average case in the early stages presents symptoms of bronchitis, either with or without emphysema, the more marked conditions, such as bronchiectasis and abscess, being usually found in those cases of long standing with excessive deposits in the lungs. When the particles inhaled have not led to any change in the bronchi or lungs, examination of the chest may be negative. The only symptom which is at all characteristic or peculiar to pneumoconiosis is that due to the presence of the foreign material in the sputum. The sputum may be black from the presence of coal-dust, soot, charcoal, or minerals, or it may contain finely divided particles of stone, metal, or the various organic substances responsible for the condition. A microscopic examination of the sputum is essential for the recognition of the foreign particles, which may be free or included within leucocytes or epithelial cells. Microscopic chemical tests may be necessary to identify absolutely the various metallic dusts, coal, or stone particles.

Prevention of the disease by adopting such measures as will prevent the inhalation of the dusts is the procedure which promises much, if it is carried out carefully and intelligently. The wearing of respiratory masks, mechanical suction devices in the workshop, or such improvements in various manufacturing processes as will obviate the necessity of exposing the workman to the dusts, are being more and more generally adopted in the large plants. The economic factors involved have interfered with their more general adoption, although many states have enacted legislation which make certain protective measures compulsory.

As the secondary infections incident to these dust diseases play such an extremely important rôle in the production of the pathologic changes in the bronchi and lungs, it is extremely important that the general hygiene of such workshops where dust is prevalent should be maintained at a very high level. Sunshine, fresh air, and cleanliness in the workrooms will go far toward overcoming the development of disease from the inhalation of dusts.

### TREATMENT.

The treatment of the condition other than by the removal of the patient from exposure to the causative factor is purely symptomatic. The prevention of further inhalation of dust is absolutely essential, and while it usually entails change of occupation, this is not necessarily always true. Where the irritation from the dust particles has given rise to secondary infection, of which tuberculosis is one of the most frequent, the treatment should be directed toward correcting or overcoming the secondary process, in addition to the removal of the primary cause. The treatment of such conditions as bronchitis, emphysema, bronchiectasis, induration, and abscesses are considered in detail in the sections dealing with these conditions, and the medical care of these processes when resulting from dust-inhalation does not differ in any way from that which is indicated when dust is not a factor.

There is no method of treatment available at the present time for the actual removal of the particles of foreign material from the lungs and bronchi. The majority of cases are mater-

ially benefited by improvement of their general health resulting from such measures as have been recommended for tuberculosis.

### PULMONARY SYPHILIS.

The pulmonary manifestations of hereditary syphilis are chiefly of interest to the pathologist. This is especially true when the lesion in the lung takes the form of "white pneumonia," which only occurs in its true form in stillborn children or in those living but a short time, usually being accompanied by a marked evidence of a general luetic infection. Gummata and interstitial pneumonic processes also may occur in the congenital form of syphilis, the latter rarely being a single process, for more commonly it is associated with one of the other manifestations of this disease. The only clinical interest attached to the various ways in which syphilis may affect the lungs in the congenital form of the disease lies in the possibility of pulmonary involvement in an infant showing evidence of a systemic infection with this disease. In the acquired form, on the other hand, the syphilitic processes of the lungs are of considerable clinical importance, as they may be the only evidence of the general infection, and upon their recognition depends the establishment of a correct diagnosis, with its definite indication of the appropriate treatment.

A form of bronchial catarrh described by some of the early writers was believed to be syphilitic in its nature, occurring during the secondary stage of the disease. With this one possible exception, the pulmonary manifestations of acquired syphilis are an accompaniment of the tertiary stage. According to numerous researches, the lungs are very rarely the seat of syphilitic disease, and while gummata or ulceration of the trachea or bronchi may occur more frequently than actual disease of the lungs, even these are far from common findings. Stenosis of the trachea or bronchi may result from the contractions resulting from syphilitic infections.

The *gummata* which develop in the lung vary in size from minute nodules to masses the size of a walnut, and are usually located near the pulmonary root, although they may be found in any part of the lung, the apex being the least frequent portion

affected. In association with the gummata there may be frequently present a *diffuse fibroid induration* of the lung. In the early stages gummata of the lungs have been described as yellowish in color, soft, irregular, and surrounded by a zone of pale red or grayish tissue, later becoming reddish, gray, yellow, or white. The gumma may undergo fatty degeneration, connective tissue formation, or even caseation. The formation of a capsule as a result of inflammatory changes in the surrounding tissue, and the evacuation of the necrotic tissue resulting in cavity formation has been described, but such changes are exceedingly rare. The tough connective tissue scars occasionally found in the lungs are believed to be in some instances a manifestation of syphilitic disease.

Microscopic examination reveals connective tissue cell proliferation in the interlobular, perivascular or peribronchial tissue, or the tissue may be so necrotic as to make it impossible to recognize the different elements. The alveoli and alveolar walls in the region immediately adjacent to the gumma contain an excess of proliferated epithelial cells, and bands of connective tissue may extend outward from the mass into the surrounding tissues. Marked changes usually occur in the blood-vessels, with thickening of the walls, the adventitia being mainly affected, with occasional changes in the media, or even in the intima. While the *Spirochæta pallida* has been demonstrated in acquired syphilitic pulmonary lesions, its detection presents many serious technical difficulties. Care must be exercised to avoid mistaking other spirochætæ which may be present in the lung for the true *Spirochæta pallida*.

Diffuse fibroid induration of the lung, occurring in association with gummata or independently, is believed to develop as a manifestation of syphilis. The process may involve parts or the whole of one lung, being usually most marked at the root. As so frequently happens in the presence of any fibroid changes in the lung, bronchiectasis may result from this form of syphilitic disease. Some question exists as to whether this diffuse fibroid induration should be considered definitely as a syphilitic process, the microscopic appearance not being distinctive. The entire problem of syphilitic disease of the lung is one which is far from being settled, some writers believing that pulmonary lesions which may be manifested clinically is



a relatively common disorder, others maintaining that it is extremely rare.

The *symptoms* of syphilis of the lung are in no way characteristic, being dependent upon the changes produced in the surrounding tissues, and not upon the lues, *per se*. Stenosis or dilatation of the bronchi may result, or possibly a combination of both conditions, with all the manifestations of bronchiectasis and, possibly, cavity formation. The rôle of syphilis in the production of fibroid changes in the lungs, either the generalized or the circumscribed form, has of recent years attracted considerable attention, especially the possibility of its causing certain of the fibroid changes at the apex. In patients presenting evidence of localized fibrosis, even at the apex of the lung, in which tubercle bacilli have been absent persistently for a long period of time, it may be well to bear in mind the possibility of syphilis as a causal factor. So little is definitely known of the various ways in which syphilis may affect the lungs, that this possibility should be considered in studying any obscure pulmonary process. While the symptoms of pulmonary syphilis are not definite or distinctive, there are certain clinical features of syphilitic disease of the lungs which should suggest its potential bearing in the questionable case. In any patient with a disease of the lung which has the general appearance of pulmonary tuberculosis, but in which tubercle bacilli are persistently absent from the sputum, when the other possible etiologic factors have been eliminated (actinomycosis, etc.), syphilis is to be suspected as the cause. Especially is this true when the general nutrition is comparatively well maintained, and when there is no elevation of temperature, or when this is subnormal. Furthermore, the dyspnea is out of all proportion to the extent of the lesion, and their distribution is atypical, especially when located in the middle third of the lung, with the apices free. It has also been stated that in pulmonary syphilis hemoptysis is very rare, and that frequently a striking feature in the early stages is the absence of moisture, as evidenced by the absence of râles. The general indications just mentioned refer principally to gummata of the lung, but when the pulmonary process produces such changes as result in circumscribed bronchiectasis, pulmonary cirrhosis,



or abscess, the etiologic relation of syphilis is not so definite. The majority of cases of pulmonary syphilis resemble pulmonary tuberculosis or tumor of the lung. The use of the Wassermann reaction has been of such material assistance in diagnosing the presence of a luetic infection, and it is so universally employed, that a word of caution may not be out of place.

When evident disease of the lung is present a positive Wassermann reaction must not be taken as conclusive evidence that the pulmonary process is syphilitic. There is no reason why a syphilitic patient may not develop tuberculosis or any other disease of the lungs. A positive Wassermann reaction, however, in a person suffering from disease of the respiratory tract, in whom every possible means of determining the actual cause of the lesion has failed to furnish any definite information, is sufficient evidence upon which to apply the therapeutic test. Even if the pulmonary lesion should not be due to syphilis, anyone suffering from a disease of the lung who shows a strongly positive Wassermann reaction should receive antisyphilitic treatment, regardless of the nature of pulmonary condition.

Cases have been reported in which syphilis was presumably responsible for the development of bronchopneumonia and a progressive destructive process in the lungs (syphilitic phthisis). These are extremely rare, and while some writers do not believe that the evidence of syphilis as an etiologic factor in the pulmonary process has been absolutely conclusive, the general tendency has been to accept them as recognized syphilitic lesions.

The *treatment* of syphilis of the respiratory tract does not differ from that of the infection in general. The administration alternately of the protiodide of mercury and one of the salvarsan preparations intravenously probably gives the best results. The general health and strength of the patient usually requires building up, such measures as have been recommended for this purpose in pulmonary tuberculosis being the most satisfactory.

The results of treatment vary with the amount of destruction of normal relations resulting from syphilitic disease, many of such processes being accompanied by changes in the

surrounding tissues, which, while dependent upon the presence of the syphilitic disease, are not of themselves actually luetic. The scarring consequent to the healing of gummata may result in such changes in the lungs or bronchi that special treatment will be required, in addition to that directed solely toward the eradication of the syphilitic infection.

### PULMONARY ACTINOMYCOSIS.

Infection of the lungs with *Actinomyces bovis* is an uncommon clinical finding, but the recording of an increasing number of cases places it among the diseases which must be suspected in any chronic non-tuberculous pulmonary affection. The sputum of such cases should be carefully and repeatedly searched for the small nodular masses containing the club-shaped filaments, which may be clearly demonstrated by Gram's method of staining. While we know that the vegetable parasite which may cause this disease in man is identical with that causing a certain disease in cattle, the mode of infection is far from being definitely proved. Primary implication of the lung usually is the form in which it is encountered, although the lung may be infected secondarily by the extension of the disease from neighboring organs.

The changes in the lung as a result of infection with actinomyces resemble those produced by tubercle bacilli in the chronic ulcerative type of the disease. It differs from tuberculosis in affecting the lower lobes more frequently than the upper, and in the fact that there is more marked connective tissue formation. The actinomycotic process is also more likely to invade surrounding structures, such as the chest wall, with the formation of sinuses, the bronchi are practically always implicated; and it very rarely affects both lungs. In the later stages of the disease the chest wall is almost always perforated, which peculiarity should of itself indicate the nature of the process, but in the earlier stages the symptoms may be merely those of a chronic bronchitis or tumor-like invasion of the lung. There are no symptoms which can be considered characteristic of the early stages, except possibly the presence of the small granules in the sputum. These can be easily demonstrated by diluting the sputum with three or four vol-

umes of water, and agitating the mixture, after which, on standing, the characteristic granules tend to sink to the bottom of the vessel. The examination of these granules under the microscope will reveal the characteristic arrangement of the clubbed filaments in a ray-form. The study of the chest by means of the *x*-rays is of very little value in this condition, except in that it may reveal pulmonary consolidation in the deeper portions of the lung in some cases in which the clinical symptoms and signs have suggested merely a bronchitis.

The medical *treatment* is purely symptomatic, being directed chiefly toward the relief of cough, expectoration, and pain. Iodid of potassium has proved very effectual in actinomycotic disease in other parts of the body, but this drug seems of only moderate value in the pulmonary form. It has been employed in doses of 1 to 3 drams (3.8 to 11.6 Gms.) a day over a long period of time, with symptomatic relief in some instances.

Operative interference seems to promise more than medicinal treatment, and should be considered in any case in which the disease tends to progress in spite of treatment. The resection of ribs or the excision of the mass is more likely to be followed by results if performed early, before extensive invasion of surrounding structures has occurred, and when the lesion is still circumscribed and localized in an accessible portion of the lung. The *x*-ray may prove of value in determining whether or not the disease is disseminated, when considering the advisability of operating.

### PULMONARY STREPTOTHRICOSIS.

The micro-organism causing this condition resembles in many ways that of actinomycosis, and many of the earlier cases reported have been due to a confusion of the two. The micro-organisms described as streptothrix probably include several species of the one group, and are characterized by the formation of slender filaments, either free or forming a loose net-work, which may occasionally show true branching. They are Gram-positive, and after staining with carbol-fuchsin are resistant to the action of acids, retaining their color to a variable degree, some of them being strongly acid-fast. The

absence of clubbing and ray-like arrangement of the mycelia, and the acid-fast qualities serve to distinguish the streptothrix from actinomycetes.

The resulting pulmonary and pleural adhesions very closely resemble those occurring in actinomycosis and in certain forms of pulmonary tuberculosis, usually showing an infiltration, with very little breaking down and no caseation, the lesions resembling those consequent to bronchiectasis, bronchopneumonia, induration, abscess, and gangrene. The pleura is frequently affected, with the formation of plastic or serous exudate, and empyema. The chest wall may become invaded, as has been described as occurring in actinomycotic infection.

The symptoms vary with the gross pathologic lesions in the lungs, resembling those due to tuberculosis, bronchopneumonia, fibroid disease, bronchiectasis, abscess or gangrene, and the majority of cases probably are mistaken for chronic pulmonary tuberculosis, malignant disease, pulmonary syphilis, or actinomycosis.

There is no specific *treatment* for streptothricosis. Attention to the general health, as suggested for the treatment of tuberculosis is advisable. The various pulmonary changes which develop should receive the treatment outlined for such conditions under bronchiectasis, abscess, gangrene, pleurisy, etc. Iodid of potassium and Fowler's solution may be tried internally, but the favorable effects ascribed to their use probably have little foundation in fact.

## PULMONARY NEOPLASMS.

The benign tumors of the lungs possess no peculiar clinical interest, being merely pathologic curiosities as occasional unexpected findings at autopsy. Malignant tumors of the lungs are not uncommon, and while usually secondary to a growth in some other part of the body, occasionally may be primary. The growth may arise in the peripheral portions of the lung or near the root, the physical signs being dependent upon the size and location of the tumor mass or masses. As the process in the lungs may consist of solitary nodules which seem to bear a close relation to the bronchial system, or a diffuse,



irregular invasion of the parenchyma of the lung, one may readily see that the physical signs may be very varied. This lack of regularity in the physical signs of pulmonary neoplasms should direct attention to the possibility of malignant disease, search being made for the primary focus of the process in some other part of the body, although, as previously stated, the pulmonary lesion may be primary.

The *symptoms* are by no means characteristic, the cough and expectoration, loss of weight and strength, not being peculiar to malignant disease. The dyspnea is usually severe, being frequently out of all proportion to the amount of lung invasion, as determined by examination, and in obscure disease of the chest the occurrence of this symptom should suggest malignant disease as one of the possibilities. Carcinoma or sarcoma of the lungs may be present to quite a considerable extent without any physical signs on auscultation, the only manifestation being dullness on percussion and diminished expansion of the affected region; or localized râles, with suppression of breath- and voice- sounds may be present. When situated near the bronchi, the tumor masses may give rise to pressure-symptoms, such as stridor, hard, unproductive cough, and dyspnea.

The *diagnosis* of malignant disease of the lung is attended by many difficulties, as the distribution of the lesions and the form they may take are not characteristic. The absence of any etiologic factor which might be discovered in the blood or sputum adds to the difficulty of the problem. Irregular, localized areas of dullness, with suppression of breath- and voice- sounds, and diminished expansion, might suggest the presence of malignant disease in the peripheral portions of the lungs, especially when occurring in a patient past middle life, who shows progressive loss of weight, severe dyspnea, and blood-tinged sputum; in such cases fever may or may not be noted. When the root of the lung is the seat of a tumor, in addition to the pressure-symptoms mentioned, there may be evidence of a mediastinal growth from an extension of the pulmonary disease. The presence of a tumor-mass in the mediastinum may be suspected from the appearance of symptoms such as dullness over the sternum, extending to either side, dullness over the upper dorsal spines posteriorly,



atypical murmurs due to pressure on the vessels, irregularities of the radial pulses, tracheal tug, pulsation over the upper anterior chest, dilatation of the superficial veins, edema of the chest wall, cyanosis, and cardiac displacement. The sputum is not characteristic, being mucoid, mucopurulent, or purulent, and at times mixed with blood. Hemoptysis also may occur as a complication. Microscopic examination may show the presence of small tumor-masses, although they are usually so decomposed or disintegrated that the cellular and morphologic character is hard to determine. Some writers lay stress upon the diagnostic value of numerous isolated cells or cell clusters in the sputum, and upon the presence of refractive spherical bodies with coarse or fine fatty granules.

When the bronchi are invaded a positive diagnosis occasionally may be made by means of the bronchoscope, and in every case the *x*-ray should be employed, if possible, as it may give information of considerable value in many cases; the radiographic findings may be at times misleading, if great care is not exercised in interpreting the stereoscopic plates.

Malignant disease of the lung is not amenable to *treatment*, and the most that can be done is to alleviate the symptoms. Recourse must be had to morphin or some derivative of opium, to render the patient as comfortable as possible. Treatment by the *x*-rays may be employed, but in such a deep-seated process not very much can be expected from their use. With our present means of examination, it is hardly possible to detect the presence of these processes in the lung at a time when they are amenable to surgical interference, the tendency for the growths in the lung to be rather widely disseminated usually contraindicating the employment of surgical measures.

## ECHINOCOCCUS DISEASE OF THE LUNGS.

This relatively rare disease in man represents the larval stage in the life cycle of the *Tenia echinococcus*, which inhabits the intestinal canal of dogs and other closely related animals. The most common intermediate hosts are sheep, cattle, and swine, and the cases in which man serves in this capacity are apparently accidental and rare occurrences. The source of the infection in human beings usually is dogs which have been

used for herding sheep, the ova from the feces of the dog gaining entrance to the stomach by means of contaminated food or water, or by hands soiled in petting or caring for the animals. The embryo develops in the stomach, and may bore its way into different parts of the body by means of the hooklets with which it is armed. The path taken by the embryo in its passage from the stomach to the lungs is not clear, the fact remaining that it may appear in the lung as a primary disease. The embryos gradually develop within the lung with the formation of a cyst, with a thick, usually laminated wall, within which is the germinal layer surrounding the cavity containing fluid. From this germinal layer heads (scolices) develop, further growth leading to the formation of cysts within the mother-cyst.

More rarely secondary pulmonary cysts, may develop from extension of the disease in the lung, or in some adjacent organ or part of the body. The rupture of hepatic cysts through the diaphragm may cause invasion of the lung. The cysts may occur singly in the lung, or they may be multiple, may involve one or both lungs, or the pulmonary invasion may be part of a fairly general invasion of different portions of the body.

Surrounding the thick elastic cuticle of the parasites a connective tissue capsule of varying thickness develops between the cyst and the surrounding lung tissue, in which chronic interstitial changes are prone to occur. Perforation of the cysts frequently takes place, with evacuation of their contents through the bronchi, or, more rarely, into the pleura, pericardium, spinal canal, or abdominal cavity. Suppuration may occur with the formation of a closed or open abscess cavity.

The cysts may remain latent in the lungs for months, or even years, without producing symptoms. The symptoms which commonly accompany the disease are cough, dyspnea, pain, and hemoptysis, the last-named being relatively a very frequent symptom, sometimes appearing even early in the disease, and occasionally being severe enough to cause death. Physical signs may be completely absent in small centrally located cysts. When large and unruptured, there may be unilateral diminution of the inspiratory excursion of the

chest, with possibly a localized prominence, sharply defined circular areas of dullness or flatness, absent vocal fremitus and resonance, diminished breath-sounds, and displacement of the heart, liver, and spleen.

Rupture of the cyst may be accompanied by hemoptysis and evacuation of the cyst contents through the bronchi, or by sudden pain and a collection of fluid in the pleura. The rupture of the cysts is frequently accompanied by an attack of urticaria, the occurrence of which may suggest the possibility of this condition being responsible for the pulmonary signs and symptoms in obscure cases. The cyst contents can be recognized by the finding of small pieces of the laminated cuticle or by the presence of hooklets, in a fluid containing considerable sodium chlorid and very little albumin. Echinococcus disease is one of the numerous causes of eosinophilia.

The  $x$ -rays are of the greatest importance in the detection of this disease, especially in the early stages. The cysts may be recognized by their sharply defined, well-rounded shadows of uniform density. Precipitin and complement fixation tests of the blood-serum have been employed for the detection of echinococcus disease.

*Treatment* of the disease is purely surgical, consisting of the excision of the cyst *without rupture*. Removal of the contents of the cyst by thoracentesis, either with or without the injection of such substances as iodine or carbolic acid, is attended by considerable danger, and should never be employed.

*Blastomycosis*, *aspergillosis*, and *distomatosis* are rare pulmonary infections in the United States, but these conditions must be considered as possibilities in obscure pulmonary conditions. Their *treatment* is purely symptomatic, as there has been no specific mode of therapy yet discovered. Their recognition depends entirely upon detecting in the sputum the characteristic micro-organisms of the disease in question.

## PLEURAL FLUIDS.

The fluids arising within the pleural space have been the subject of careful study and investigation for many years, in the hope of obtaining data of sufficient constancy to be of value in determining the nature of the morbid process respon-

sible for the effusion in the individual case. The problem is complicated by the fact that in a great many pleural effusions the process is not simple, but, on the contrary, referable to several different conditions. Thus, an exudate may have added to it a certain amount of transudate, or a pleurisy, originally purely tuberculous, may become contaminated with some secondary infection. The confusion caused by the conflicting results reported by different observers in studying similar conditions is probably due to the fact that the effusion studied did not conform to any one distinct type. It is extremely important that the information obtained by the study of any given effusion should be considered only in its relation to the clinical findings in the individual case. With the exception of a few positive diagnostic findings, the laboratory study of the pleural effusions is mainly of value in confirming a diagnosis, or in suggesting certain etiologic possibilities.

The chief object in such findings has been to obtain such information of value in deciding whether such a collection of fluid is an exudate or a transudate, and, if the former, the nature of the disease responsible for its accumulation. When the effusion is secondary, it is not uncommon for the primary disease to be so obvious that the study of the fluid is unnecessary. In primary pleural effusions, or in those secondary effusions in which the primary disease is concealed, the study of the fluid may prove of the greatest importance.

The gross appearance of the fluid usually indicates whether it is serous, serofibrinous, fibrinopurulent, purulent, chylous, or hemorrhagic. A microscopic or cultural study is necessary to determine the presence of bacteria or small quantities of pus, blood, chyle, or fibrin.

As a rule, transudates have a low specific gravity (1.010 to 1.015), with an amount of albumin ranging from a trace to as high as 3 per cent. Exudates, on the other hand, have a specific gravity of 1.018, or higher, and an albumin content of 4 per cent., or greater. The exudates show a fairly abundant precipitate when acetic acid is added to the fluid, and usually show a tendency to coagulate rapidly. In cases close to the dividing-line between exudate and transudate the albumin should be estimated by some of the more delicate methods, as the customary Esbach's test is not sufficiently accurate.



*Cytology.* The cellular elements in the effusion have been extensively studied, in the hope that their general character and the proportion of the various types of cells present would prove of value in differentiating effusions due to different diseases. The cells are obtained from the sediment of the citrated, centrifugalized fluid, mounted on a slide, and stained by Wright's blood-stain. A differential count is made of the leucocytes, a record being made of the number of neutrophiles, basophiles, lymphocytes, and endothelial cells in the same manner as in a blood-examination. The leucocytes found in the effusion not infrequently show degenerative changes and pigmentation, which makes the process of counting at times somewhat difficult. Occasionally cells are encountered in which it is hard to determine whether they are polymorphonuclear or mononuclear, and these atypical forms are best grouped under a separate heading, such as "cells of uncertain type." The differentiation between lymphocytes and endothelial cells also may be extremely difficult in a small proportion of the cells.

The original observations on the leucocytic counts in pleural effusions resulted in the statement that when the polymorphonuclear cells predominated the effusion was of an infectious origin, when the lymphocytes were in excess it indicated a tuberculous process, and when the endothelial cells were in the majority, especially if arranged in sheets or plaques, the effusion was of a mechanical type.

While the various leucocytic formulæ may be of value as suggesting various causes for the presence of fluid, they must not be considered as absolutely diagnostic, more recent observations having shown that the leucocytic picture varies, not only in different processes, but also in various stages of the same process. It has been shown that in the tuberculous effusions a temporary increase of the polymorphonuclear elements may be found in the early stages, and that a secondary infection may modify the numerical relation of the cells. In transudates of long standing the lymphocytes may be in excess instead of the endothelial cells, and an excess of lymphocytes occasionally may be found in non-tuberculous exudates. The effusions due to malignant disease usually show an excess of endothelial cells. The polymorphonuclear predominance



commonly present in effusions due to the pneumococcus, streptococcus, or staphylococcus is probably the most constant of the various leucocytic formulæ.

*Bacteriology.* For the detection of pneumococci and the ordinary pyogenic micro-organisms, the usual methods of cultivation are sufficiently reliable. The presence of tubercle bacilli is not easy of demonstration, the cultivation of these bacteria presenting so many difficulties as to render this method of investigation practically useless. The microscopic examination or animal inoculation of the sediment obtained by centrifugalization of large quantities of the fluid are the most reliable methods for the determination of these micro-organisms.

The coagulation of the fluid may interfere seriously with this procedure, but usually it may be overcome by the addition of sodium fluoride or sodium citrate to the freshly drawn serum, or by the digestion of the coagulum after the method of Jousset. Inoculation of animals for the detection of the tubercle bacillus must be very carefully performed to be of any value, and it is of value only when the result is positive, as negative findings do not absolutely exclude the possibility of a tuberculous lesion.

### ACUTE FIBRINOUS PLEURITIS.

The treatment of acute fibrinous pleuritis necessitates a knowledge of the underlying causes responsible for a development of this process, for it must be considered as purely a secondary process in every instance, even if in certain cases the factor responsible for its presence cannot be discovered. The so-called primary cases constitute a very small proportion of the number encountered, in which the etiologic factor cannot be actually demonstrated or strongly suspected. From a practical standpoint, then, the first principle in the management of a case of acute fibrinous pleuritis consists in the relief of the patient; the second, which is equally important, is the employment of every means in our possession to determine the actual or probable condition responsible for the pleural inflammation. While exposure and alcoholism are possible causes, by far the largest number of cases are due

to tuberculosis, so that any patient presenting signs of this disease without evident cause being detected should be viewed as a probable case of tuberculosis, and every means employed to obtain information as to the possibility of such an infection by a study of the family and previous history, symptoms, and physical examination, including such clinical and laboratory tests as may prove of value. It must be borne in mind that, while the majority of cases are due to tuberculosis of the lungs this is not necessarily always the case, for the pleural inflammation may result from a general infection with tuberculosis, or as a result of tuberculosis of some organ in the body other than the lung. The pleural condition may precede the pulmonary, at least so far as one may determine by the clinical means in our possession at the present time.

The processes other than tuberculosis which may be accompanied by or be responsible for, the development of inflammation of the pleura, include all the general infections and bacterial disease of any part of the body, or it may accompany the terminal infections of one of the chronic, asthenic diseases. The diseases of the lungs, such as lobar or bronchopneumonia, abscess, gangrene, and infarction, naturally account for many of the cases, although bronchitis and bronchiectasis also may bear a causal relation. Pleuritis may complicate such conditions as acute or chronic endocarditis, tonsillitis, arthritis, pyorrhea alveolaris, pericarditis, typhoid fever, urethritis, and pelvic sepsis. Trauma also may rarely be responsible for the development of the disease.

The pleura at the seat of inflammation is dull and opaque, the normal glistening appearance being absent, and the surface coarsely or finely granular and usually more or less thickened. The color is grayish-white or reddened, occasionally being of a dark-red color, due to the extravasation of blood. Occasionally the membrane is thickened to a marked degree, or covered with a thick, shaggy, plastic exudate. The entire surface of one lung may share in the process, or only small localized patches; a small amount of fluid in excess of the normal is almost always present. Microscopically, the lymph- and blood- vessels are dilated, the subserous tissue is swollen and contains numerous polymorphonuclear leucocytes, and the surface shows degeneration and desquamation,

with a covering of fibrin containing serous exudate and leucocytes. This layer of exudate may undergo fatty changes and absorption, or the round cell infiltration may organize and form connective tissue, with possibly complete adhesion of the pleural surfaces.

The *symptoms* may be modified or obscured by the associated disease, but in the majority of instances the pleural attacks come on suddenly, with little or no premonitory signs, slight elevation of temperature, and, rarely, with an initial chill. The most characteristic and constant symptom is pain, which is sharp, cutting, or stabbing in character, or occasionally dull and dragging. The pain may be constant or only present on deep breathing or coughing. It is usually localized to the anterior axillary region over the lower portion of the chest, but may extend posteriorly, or radiate toward the shoulders. In rare instances the pain may be referred to the abdominal cavity, thus naturally directing attention to that part rather than to the chest, and hence frequently leading to a mistake in diagnosis, especially when the pain is accompanied by tenderness and rigidity of the abdominal muscles. Rapid, shallow breathing may be present, from an unconscious effort on the part of the patient to limit the respiratory excursion, or, in rare instances, true dyspnea may occur. While cough is usually present and is believed to be actually pleuritic, the difficulty in excluding the other possible causes of cough is extremely difficult, especially as the pleurisy is so frequently associated with some other bronchopulmonary disease.

*Physical examination* shows the patient in a position which tends to limit the expansion of the affected side, the shoulder being depressed and the arm closely held against the ribs. The decubitus varies in different cases, some lying on the affected side, others on the unaffected. There may be moderately suppressed breath-sounds, and slight impairment on percussion, with decreased vocal fremitus over the affected area, but usually these signs are lacking. The characteristic sign is the presence of a friction-rub, which sounds like the creaking of leather, is synchronous with inspiration, becoming intensified toward the end of the inspiratory phase, and is unaffected by cough. Certain types of

râles may simulate a friction-rub, but these may be distinguished by their tendency to disappear or to become intensified by cough, by their being less regular or constant, and unaccompanied by severe pain. The grating, rumbling sound heard in many normal individuals over the upper half of the posterior thoracic region when the arms are folded across the chest is due to crepitus in the shoulder joint. There is some doubt as to whether the fine crepitation occasionally heard in patients suffering with localized pain in the chest is due to pleuritis or to moisture in the pulmonary tissues immediately beneath the pleura. When the diaphragmatic surface of the pleura is affected, the diagnosis may be extremely difficult owing to the complete absence of friction-sounds, and to the fact that the pain may be referred to the upper chest or abdomen, depending upon the portion of the diaphragm which is inflamed. Gross changes in the lungs may obscure the signs of pleurisy.

#### TREATMENT.

The treatment consists primarily in the adoption of measures to ensure relief of the pain, which may be of agonizing severity. When the pain is not very severe, local applications of heat, and the administration of heroin or codein, or possibly a hypodermic injection of morphin may give a certain amount of comfort, but there is nothing which meets the requirements so well as strapping the affected side with adhesive plaster, the zinc oxid preparation being the best.

The strips of adhesive, about 2 inches (5.08 cm.), should be cut beforehand the required length, six strips usually being sufficient. Preferably the patient should be in the upright position, with the shoulder of the unaffected side braced against an immovable object, such as a wall; the first strip attached posteriorly, so that it extends about 2 inches (5.08 cm.) beyond the spinous processes on the unaffected side, just above the level of the lower costal margin. At the end of a forced expiration, the adhesive strip is forcibly applied around the chest, in a slightly downward direction, tension being made by manual traction of the anterior end of the strip. As it is brought in apposition to the chest, firm traction must be kept up until the strap has completely surrounded the



affected side of the chest, with the end passed about 2 or 3 inches (5.08 to 7.62 cm.) beyond the anterior median line, where it must be held for a few seconds until it adheres tightly to the skin. The second strip is placed in the same manner just above the first, and overlapping about one-third of its width. This is continued with other straps until the chest has been covered up to the axillary folds, care being taken to see

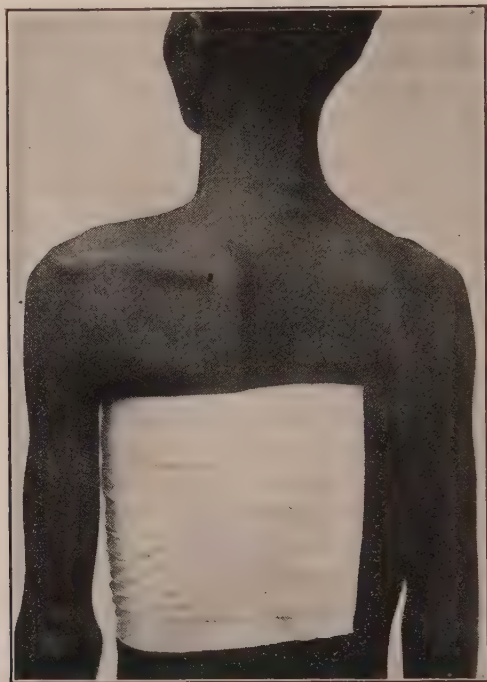


Fig. 23.—Strapping of the chest in acute pleurisy.

that they are placed smoothly and evenly. In a woman with generous breasts it may be necessary to pass the anterior ends of the few upper strips forward and upward, and the lower ones slightly downward and forward, so as to avoid covering the uneven prominence of the mammary gland. The anterior and posterior extremities of the strips should be covered with another strip of adhesive running vertically to prevent their curling or becoming loose and slipping. Strapping of the chest may seem a very simple matter to describe at such



length, but it is absolutely useless, and may be very distressing to the patient, if not performed correctly. It is extremely important that the ends of the strips should pass well beyond the anterior and posterior median lines, overlapping 2 or 3 inches (5.08 or 7.62 cm.) on the unaffected side, and considerable force is to be used in their application, if fixation of the affected chest is to be assured.

Care must be used in removing the adhesive, as this procedure usually is accompanied by considerable pain, to which patients ordinarily do not object, if it is not unduly prolonged. Inasmuch as the skin is partly denuded by the process, the use of turpentine or ether for loosening the plaster is very objectionable. Soaking the strips overnight with cottonseed oil or olive oil will often facilitate their removal. Freeing both extremities of the strips first, and then pulling on them quickly and firmly, will usually be found more satisfactory than attempting to loosen them for only a short distance at a time. After they are removed the partly denuded skin should be quickly coated with a bland, soothing, heavy ointment, such as zinc oxide, as even the contact with the air may be very painful.

Not only rest of the lung is important, but rest in bed should be insisted upon, the patient being allowed to assume the position which seems to give the greatest amount of comfort, although it is perhaps preferable to have him lie on the affected side.

Search should be made for a possible source of infection, which when found should receive appropriate treatment. In the absence of any evident or probable cause, as stated above, the case should be considered as probably tuberculosis, and in the presence of any strongly suggestive history, symptoms or signs, it is to be treated as such. In such an event it is advisable to inform the patient that tuberculosis is in all likelihood the cause of the trouble, and to warn of the importance of maintaining a rational mode of life. Any symptoms such as loss of weight, cough, digestive disturbances, temperature, hemoptysis, should be heeded, and a careful physical examination made upon the development of any sign suggestive of phthisis. Before such patients are permitted to resume their ordinary occupation, their general health should be built up

A.



B.



Fig. 24.—Showing method of determining the relative expansibility of the lower portions of the chest in patient with old left-sided pleurisy. A, during expiration; and B, at the end of inspiration. Note the difference in distance from spinous processes of the right and left thumbs.

to a better state than before the attack, even if sanatorium treatment is necessary to accomplish the desired result.

Where the fibrinous exudate has been extensive, the acute attack may be followed by a diminution of expansion on the affected side, varying in amount from so slight a decrease as to be hardly detectable to the virtual abolition of respiratory movements. When the restriction is serious, various measures may be employed to increase the expansion of the lung, such as breathing exercises and forced expiration. Some care must be used in resorting to breathing exercises absolutely to exclude the possibility of any active pulmonary tuberculosis, or disastrous results may follow, with possibly rapid extension

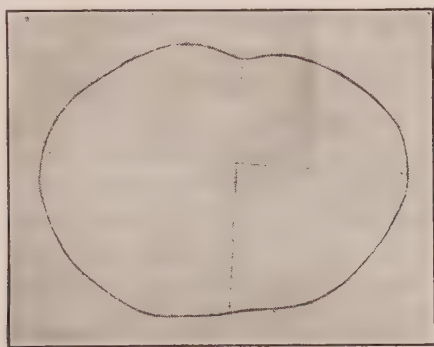


Fig. 25.—Tracing of lower portion of chest (level of 5th rib) in patient with old left-sided pleurisy. Note the marked difference between the size of the two sides of the chest.

of the pulmonary process. When slight or moderate restriction is present, it is much safer to dispense with deep-breathing exercises, and to warn the patient of the dangers attending their use. The operative treatment suggested for the cases with dense, firm, organized exudate, such as decortication, cannot be recommended, despite the fact that fibrosis of the lung may possibly develop as a result of such pleural changes.

### SEROFIBRINOUS PLEURITIS.

A small proportion of serous pleural effusions appear to be primary, that is, no definite cause for the development of the effusion can be determined. The disease which is most fre-

quently responsible for the process is tuberculosis, and what was said in regard to the relation between this disease and the fibrinous type of pleurisy is applicable here.

Among the non-tuberculous effusions the most frequent cause is the pneumococcus, although streptococci and other micro-organisms may at times be responsible. Effusions of this type are not an uncommon sequel of pneumonia, in which there is a decided tendency for them to become purulent. Other diseases in which serous pleural effusions may occur are rheumatism, endocarditis, typhoid fever, trauma, and infections of various parts of the body.

In addition to the changes found in the simple fibrinous type of pleuritis, there is a variable amount of fluid in the dependent portion of the pleural space. Above the level of the fluid the pleuræ may be adherent, united by fibrous bands, or merely covered with a small amount of plastic exudate, the two surfaces being easily separated. The pleura may show miliary tubercles on gross or microscopic examinations in cases in which the tubercle bacillus is the etiological factor. The fluid is usually pale amber in color and may contain fibrin in the form of thin, thread-like or irregular, thick, dense clots. The dividing-line between serofibrinous fluids which are cloudy from the admixture of numerous cells and those containing an admixture of pus is by no means definite and fixed, the two types frequently shading into one another. The study of the pleural fluids is considered in more detail in a separate section (see Pleural Fluids, p. 499).

While small amounts of fluid may exist with a negative intrapleural pressure, with increasing amounts this usually becomes positive, depending upon the amount of serous exudate and the presence or absence of pleural adhesions or pulmonary disease. The lung becomes contracted as a result of the presence of the fluid in massive effusions, and it may be compressed into a small mass lying against the vertebra in the upper posterior part of the thoracic cavity, more or less completely devoid of air and blood. When the lung is not the seat of marked inflammatory changes, even after having been compressed for a considerable length of time, still it is capable of re-expansion. This is not so likely if the fluid contains much fibrin or cellular exudate, as adhesions are more

likely to form and prevent re-expansion. The purulent effusions are even less likely to be followed by complete re-expansions.

The onset of the disease varies very much, for while some cases arise suddenly, with all signs of an acute illness, in others the onset may be so insidious as to pass unnoticed by the patient, being detected only on routine examination. It is not a very uncommon experience to have an individual come for examination on account of slight dyspnea and to discover an entirely unsuspected pleural effusion, perhaps so massive as to reach nearly to the level of the clavicle.

The *diagnosis* does not, as a rule, present any great difficulties in the average case, although we must admit that it is only possible to recognize effusions clinically after they become fairly large. In the presence of extensive disease of the lung, or adhesions in the pleural space, the diagnosis of a small effusion may be difficult.

Pain may be present in the early stages, but it usually decreases as the level of the fluid rises. A short, dry cough in uncomplicated cases is very common, with a variable elevation of temperature and an increased pulse-rate.

The physical signs vary with the amount of fluid present; when slight, the appearance of the patient and inspection of the chest may not differ from that described under Fibrinous Pleurisy (*q.v.*). As the amount of fluid increases, there is more and more dyspnea, which may be extremely severe, especially in the cases which develop rapidly.

Loss of motion, bulging of the interspaces on the affected side, and increase in their width, as determined by actual measurement, may be noted. Litten's shadow phenomenon on the affected side is usually absent, the cardiac apex-beat may be seen to be displaced, and in rare cases there is pulsation of the chest wall on the affected side.

Palpation, in addition to confirming the information obtained by inspection, may reveal downward displacement of the liver or spleen. Tactile fremitus is absent in practically every case, and constitutes a very valuable diagnostic sign of fluid. Percussion shows normal resonance (in lungs not diseased) over the upper portion of the chest, and flatness at the base; between the two there is usually a zone over which



the resonance has a tympanitic quality (Skoda's resonance). In some cases this tympanitic area extends to the apex on the affected side. When the fluid is of any considerable amount the sound at the base is flat, and usually accompanied by a distinct sense of resistance to the pleximeter finger during percussion. Above the level of the flatness there is usually a narrow zone of dullness, which becomes wider, or is only

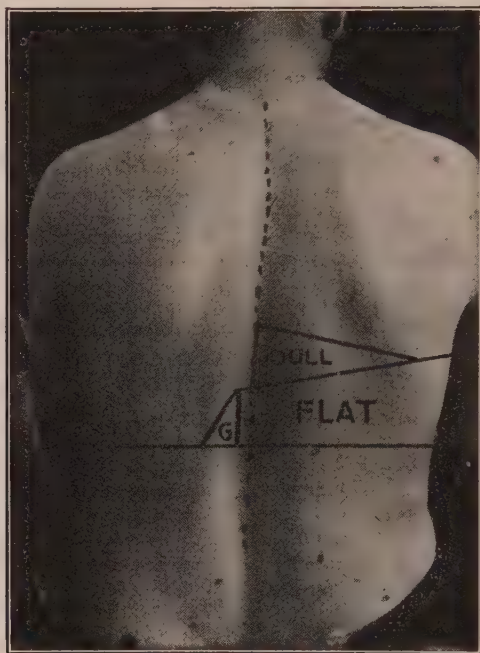


Fig. 26.—Percussion findings in right-sided pleural effusion. Note area of flatness with triangular zone of dullness immediately above. To the left of spinal flatness is the area of dullness known as Grocco's triangle (G).

noticeable posteriorly. The flatness merges below with the liver dullness when the fluid occupies the right pleural sac, but in the left-sided effusions gastric tympany may obscure the signs. In massive left-sided cases Traube's semilunar space may be obliterated. The upper level of the flatness usually forms a curved line around the chest when the patient is in the upright position, usually being highest in the axillary

region. Pleural adhesion may interfere with this characteristic curve of the upper border of flatness, and also with the shifting of the level of the fluid with change of position, which otherwise is usually present.

The flatness corresponding to the level of the fluid, can usually be elicited by percussion of the spine. A triangular area of dullness (Grocco's triangle) on the unaffected side always can be elicited in effusions which are not encapsulated, when they have attained a moderate size. The vertical side of the triangle corresponds to the flatness obtained by percussing the spinous process, the horizontal base to a continuation of the lower level of pulmonary resonance, the obliquely vertical outer side of the triangle being determined by percussing the unaffected side in a series of horizontal lines parallel to the lower border of pulmonary resonance. It is usually found to be a right-sided triangle, with a long vertical base corresponding to the spine, the short side being horizontal, and a long hypotenuse extending obliquely downward and outward from the upper level of flatness of the spine to a point  $\frac{4}{5}$  to  $2\frac{4}{5}$  inches (2 to 7 cm.) from the spine at the lower level of pulmonary resonance on the unaffected side. Occasionally the legs of the triangle are of equal length.

With displacement of the heart toward the unaffected side, percussion may be employed to confirm the findings as determined by inspection and palpation, also the downward displacement of the liver or spleen depending on the side affected. Auscultation may reveal the presence of a friction-rub above the level of the fluid, especially in the early stages when the fluid is slight in amount. The breath-sounds over the upper portion of the lung may be vesicular, suppressed, or indefinite, becoming more broncho-vesicular in character as the compression of the lung increases, or they may be inaudible in massive effusions. Just above the level of flatness the breath-sounds are frequently broncho-vesicular in character, especially posteriorly, with increased vocal resonance, which may be bleating in character (egophony), and fine, moist or crepitant râles. The area over which these signs are present corresponds to the area of dullness above the level of flatness, being usually confined to the posterior

thoracic region. Over the flat portion of the chest the breath- and voice- sounds are absent. Cases are occasionally encountered in which bronchial breathing can be heard over a pleural effusion, but the cause of this paradox has never been satisfactorily explained. The transmission of the whispered voice through an effusion (Bacelli's sign), has been claimed to indicate a serous rather than a purulent effusion, but this peculiarity is too unreliable and uncertain to be of any clinical value.

The employment of radiographs for studying the cases in which fluid is suspected frequently is of considerable value in confirming the results of physical examination, especially in those cases characterized by loculation of the fluid or by obscure physical signs. Fluoroscopic examination is not so satisfactory as the use of plates, except in a study of the diaphragmatic excursion. The cardiac and diaphragmatic displacements and the exact extent of the effusion may be definitely determined by this means of study, and the presence of pulmonary disease suggested.

The diagnosis of intrapleural fluid may be absolutely confirmed, and considerable valuable information obtained, especially in regard to the actual cause of the disease and character of the fluid, by means of exploratory puncture. This may be made painless by the injection of a small quantity of novocaine and adrenalin solution, which is placed in the syringe and injected very slowly. In addition to obtaining sufficient fluid for study, the resistance met with by the needle frequently indicates any thickening of the pleura, or increase in its density. Rarely small plugs of tissue are caught in the lumen of the needle, and may be studied microscopically, the information thus obtained in very rare instances being sufficient for an absolute diagnosis as to the nature of the pleural process.

Having determined the existence of fluid in the pleural space, there are several points in regard to which it is necessary to have additional information before one may outline the proper course of treatment.

In the first place, it is necessary to determine whether one is dealing with a pleural exudate or with a transudate, or, in other words, with a true inflammatory process of the pleura

or an osmotic transudation due to hydremia or stasis secondary to cardiac or renal disease. The presence of any disease of the heart or kidney should suggest the probability of a hydrothorax, or the general course and symptoms may suggest a pleuritis, but a physical study of the fluid is necessary to distinguish definitely between these two conditions.

The question of tuberculosis as a cause of the exudate also is extremely important, just as in the fibrinous form of pleurisy, only here we have the additional advantage of the data to be derived from a study of the fluid. A study of the cellular elements of the fluid may be of some help, although such findings are not to be absolutely depended upon. Examination of the sediment obtained by centrifugalization, digesting the fibrinous clots for the presence of tubercle bacilli, and the use of animal inoculations are absolutely conclusive, when the results are positive. Unfortunately, when the presence of tubercle bacilli cannot be demonstrated it is not possible to exclude tuberculosis as a possible cause of the process (see *Pleural Fluids*). Where no obvious cause for the development of the effusion can be detected, it would seem the wisest plan to consider it as tuberculous, unless proven to the contrary.

#### TREATMENT.

The general treatment of the secondary pleurisies must to a great extent be governed by the nature of the process responsible for the effusion. Where the primary disease is not discoverable, the patient should be treated as if suffering from tuberculosis, especially as three or four out of every ten cases of this type of effusion are likely to manifest active pulmonary tuberculosis within a few years. The patient should be warned of the danger which may result from neglected attacks of cough, temperature, or other evidence of disease which may indicate beginning pulmonary tuberculosis, and impressed with the importance of maintaining his health at the highest possible level, and of not neglecting the development of any unfavorable symptom. These patients should receive the same careful attention as one would give a frank case of phthisis, being advised of the importance of fresh air, rest, nutritious diet, and regulated exercise. (See p. 412.)



The treatment should be directed toward encouraging the absorption of the fluid by increasing the elimination of fluids from the body through the skin, kidneys, and intestines. While this method is generally recommended, it must be acknowledged that it is questionable whether any impression can be made upon a pleural effusion by increasing the excretion of water from the body, even when employed in combination with a dry diet. Small doses of potassium iodid, or syrup of the iodid of iron have been recommended as of value in hastening the absorption of pleural effusions of long standing. The salicylates in doses of 1 dram (3.8 Gms.) to 2 drams (7.7 Gms.) in the twenty-four hours are claimed to be of considerable benefit in the early stages of pleuritis.

Local applications of heat, and in some cases an ice-bag, is useful to relieve the pain and distress, and blisters applied to the chest at times appear to assist in the absorption of the effusion. The prepared cantharides plaster is the most convenient method for blistering the skin, a piece of the plaster 2 or 3 inches (5.08 or 7.62 cm.) square being applied to the skin, after carefully cleansing, at a point corresponding to the level of the fluid. The plaster should remain in contact with the skin until a blister has formed, or a marked hyperemia has developed. Should a blister fail to develop, the plaster may be removed and cloths wrung out in hot water applied, and changed at frequent intervals. By choice, the blister is allowed to remain unbroken, being merely covered with a loose gauze dressing and a bit of cotton, or the skin may be broken and boracic acid ointment applied to the surface.

The measure which is by far the most satisfactory for the removal of an effusion is aspiration. This procedure, when carefully performed is very seldom accompanied by any danger, at least with none which cannot be guarded against by the exercise of care and judgment. When an effusion into the pleural cavity is detected, the question arises as to when to aspirate, the quantity to remove at the operation, and whether any contraindications to this method of treatment are present.

Thoracentesis should be immediately resorted to if pressure-symptoms are present, such as evidence of rapidly developing cardiac failure, with rapid pulse and cyanosis, or



severe dyspnea, sufficient fluid being removed to alleviate the dangerous symptoms. If the effusion is massive, and has produced marked displacement of the viscera from their normal positions, the withdrawal of the fluid should not be delayed, even if pressure-symptoms are not present. Even when there is only a moderate amount of fluid which has shown no tendency to become absorbed under active general and local treatment, the advisability of removing some of it must be considered, as a slight reduction in the amount of the effusion may be sufficient to allow the remainder of the fluid to be absorbed.

In the face of an actual or suspected active pulmonary tuberculosis great caution must be exercised in the withdrawal of the fluid (as described under Tuberculosis). Removal by aspiration is sufficient when the fluid is clear, but when cloudy and suggestive of pus, it may be necessary to consider the advisability of an open operation, with the establishment of permanent drainage. The general symptoms of the patient, the factor responsible for the effusion, the condition of the lungs, and the character of the fluid all have to be considered in deciding upon whether it is necessary to resort to thoracotomy or not. While thoracentesis alone may cure a certain number of purulent effusions, it cannot be counted upon invariably to do so, and the dangers which attend delay in promptly draining pus in the pleura are so great that the open operation should be performed as early as possible in every case of empyema. When the fluid is merely cloudy, of moderate amount, and the patient non-toxic, simple aspiration may be tried, with the understanding that the more radical operation be performed on the first appearance of any unfavorable symptom or change in the character of the fluid.

*Thoracentesis.* The use of a trocar connected with a two-way outlet is much to be preferred to the use of a needle. The fittings should be absolutely air-tight, so as to prevent the possibility of air gaining admission to the pleural space. After entering the pleural space the fluid may be removed by siphonage or by aspiration, the latter being by far the most satisfactory. When about to perform this operation a great deal of annoyance and chagrin may be avoided by first testing out the apparatus, in order to see that the instrument is

complete, patulous, air-tight, and in perfect working order. The connections of the trocar, cannula, stylet, and stop-cock should be gone over carefully, and it is always advisable to see that the pump for exhausting the vacuum bottle has a tight-fitting plunger.

The skin having been scrubbed with soap and water, and sterilized with iodine, local anesthesia is induced by the hypodermic injections of a 4 per cent. solution of novocain, either with or without the preliminary use of an ethyl chlorid spray. The entire outfit, with the exception of the pump, should be sterilized by boiling; the glass bottle used to receive the aspirated fluid also should be sterilized, if a bacteriologic study of the fluid is intended.

The trocar is inserted in the interspace selected, and its point kept close to the upper edge of the rib, in order to avoid the intercostal vessels. Widening of the interspace may be secured by shifting the position of the body. The erect position in bed is the best, with the subject reclining upon a firm cushion upon the unaffected side, and the arm placed across the chest with the hand resting upon the corresponding shoulder. The fourth or fifth interspace in the mid-axillary line is the most convenient and satisfactory point at which to insert the trocar when the effusion is large; when small, the seventh interspace in the post-axillary region is better. The instrument is grasped firmly in the right hand, with the end resting against the palm of the hand, and the index finger pressed against the trocar about  $1\frac{1}{2}$  inches (3.81 cm.) from the point, so as to prevent too deep insertion. The trocar is thrust steadily and firmly into the pleural space, hugging the upper border of the rib, a sudden loss of resistance indicating when its point enters the effusion. The stop-cock on the lateral opening of the instrument is kept closed until the stylet has been withdrawn to its limits, and the stop-cock on the straight bar turned off. The cock leading to the rubber tubing, which should have a small section of glass tube inserted in its length near the instrument, is then turned on, and the fluid allowed to run slowly into the bottle, from which the air has been exhausted by means of the hand-pump. It is a mistake to make the negative pressure in the bottle too high, as very little is suffi-

cient to withdraw the fluid when the cannula is in the pleural space and not occluded by fibrin. The instrument should be held firmly in position, and a lookout kept for any sensation indicating that the opening had been suddenly occluded by a particle of fibrin, or that the lung was in contact with the cannula. In either event the obstruction may be removed by means of the stylet, or the position of the cannula shifted by withdrawal or lowering of the point.

When sufficient fluid has been obtained the stop-cocks should be turned off, a piece of gauze held around the trocar tightly against the opening in the skin, and the instrument quickly withdrawn. The opening in the skin should be sealed, and while occasionally a small amount of oozing may occur, this can be avoided by slightly displacing the skin upward on inserting the trocar so that when complete the opening in the muscular tissue is not in direct apposition to the opening in the skin. A small collodion and gauze dressing is usually sufficient, but a large dry-gauze dressing may be applied over the small dressing, and held in position with adhesive plaster if any oozing occurs.

During the withdrawal of the fluid the operation should be discontinued if severe cough, severe pain, symptoms of shock, hemoptysis, or dyspnea develop. Not infrequently the operation is accompanied by a slight sense of faintness or vertigo, usually relieved by having the patient lie flat in bed for a few minutes, when the paracentesis may usually be continued. At times some difficulty may be experienced in inserting the trocar on account of the narrowness of the interspaces because of the patient's unconscious resistance to the introduction of the trocar by fixation of the chest and inclining the shoulders downward toward the affected side. It must be remembered that the chest wall varies in thickness, and while the average is from  $\frac{4}{5}$  to  $1\frac{3}{5}$  inches (2 to 4 cm.), in very stout people it may be necessary to insert the trocar to a greater depth before the fluid is reached. In some cases, especially those of long standing, there may be a considerable thickening of the pleura, usually recognized by the sense of resistance to the needle. The fluid should be removed slowly, and with advantage the aspiration may be suspended for a few minutes intermittently, for if aspirated too fast unfavorable symptoms

may develop, and occasionally prove serious. From fifteen to twenty minutes should be allowed for every 34 ounces (1000 mils) of fluid withdrawn, the amount to be aspirated varying in different cases, but never exceeding 68 ounces (2000 mils). It is not necessary to evacuate small effusions completely as the small amount of serum left in the pleural space usually is readily absorbed. When the effusion is massive, it is better to complete the removal of the fluid at a subsequent operation than to remove a very large amount at one sitting.



Fig. 27.—Apparatus (Potain) for performing thoracentesis. Instead of the straight needle shown in the illustration, a needle fitted with stylet may be employed.

The danger of infecting the pleura is insignificant, if care is exercised in preparing the patient and the instruments, and in performing the operation. Attention to the fittings of the aspirator, and preventing any injury to the lung, will avoid the possibility of pneumothorax. With some instruments air may be pumped into the glass container, instead of withdrawn, if the wrong nozzle of the pump is attached to the rubber tubing, and it is well to see to this point personally, and not depend upon an inexperienced assistant to manage the pump. Potain's model is probably the most satisfactory aspirator now available (Fig. 27).

Subcutaneous emphysema occasionally develops locally or generally, but as a rule, it is not of a serious nature. Pulmonary edema and albuminous expectoration rarely occur, usually as a result of the sudden withdrawal of a large amount of fluid. Sudden death from the operation has occurred, in consequence of shock, embolism, or hemoptysis, but this accident is exceedingly rare. Care must be exercised in some cases to avoid wounding the heart or diaphragm, when the thoracic viscera have been forced to assume abnormal positions by pressure or by contraction induced by disease within the chest.

The after-care of subjects of pleural effusion includes such measures as may be necessary to meet the indications. The building up of the general health, especially when tuberculosis is suspected, and the restoration of a normal pulmonary expansion in effusions obviously not due to tuberculosis, will in the average case be the main objects of the treatment after the fluid has disappeared from the pleural space.

### PURULENT PLEURITIS.

The purulent form of pleural effusion, or empyema, is rarely a primary process, being usually secondary to some disease of the lung, pneumonia being by far the most common cause; less frequently, tuberculosis or gangrene is the primary factor. The process may extend from a neighboring organ to the pleura, arise in the course of some infectious disease, or occur as a complication of suppuration in some other part of the body. Serofibrinous pleurisy may become purulent as a result of the primary bacterial invasion, or after a later bacterial infection in an effusion previously sterile. It is more common in children, in whom the serofibrinous form is relatively infrequent. The pneumococcus, streptococcus, staphylococcus, and tubercle bacillus are the micro-organisms usually responsible for this condition, the first-mentioned being by far the most frequent cause ("metapneumonic empyema"), the other bacteria named but seldom having a direct etiologic relation.

In this type of pleuritis the pleura is usually thickened, of a grayish or yellowish color, and covered with a rough or



shaggy fibrinous or purulent exudate. The entire pleural surface of one lung may be affected, or the pathologic process may be confined to the lower portion, or the collection of pus may be walled off from the general pleural space forming what is known as *loculated empyema*. Areas of ulceration and breaking down of the parietal or visceral pleura may be present. The changes in the other organs as the result of the disease responsible for the formation of an empyema can usually be demonstrated. The lung is most frequently the seat of disease, as the large majority of empyemata develop as the result of some pathologic process in this organ.

The physical signs and symptoms are practically the same as in the serofibrinous type, except that in empyema the onset is usually more acute, and more commonly associated with chills, sweats, higher fever, and a higher pulse- and respiratory- rate. The signs of general toxemia are usually out of all proportion to the amount of fluid present. While usually rapid in development, and tending to become progressively worse, in rare instances even massive collections of pus may become encysted, with a disappearance of all evidences of marked toxemia, the purulent material occasionally becoming sterile, with the patient presenting merely such symptoms as would indicate compression of the lung. These sterile collections of pus may persist in the pleural space for months or even years, one such case having occurred in the experience of the writer which had apparently resulted from a pneumonia following an operation for some pelvic suppurative condition five years previously.

The *physical signs* are identical with those found in effusions of a serofibrinous character. The displacement of the heart, liver, and spleen is perhaps more marked in purulent effusions, as also is the bulging of the interspaces. Edema of the chest wall is more frequent in purulent than in serofibrinous effusions. When the empyema is of long standing, clubbing of the fingers may be very marked, and even in relatively acute cases changes in the finger-nails may be observed. Bacelli's signs for differentiating the two types by means of the transmission of the whispered voice is too uncertain to be depended upon (*v.s.*). In certain cases, especially in children, over a purulent effusion bronchial breathing may be distinctly

heard. Pulsation is more frequently encountered in the purulent than in serous effusions, although even the latter is a rare phenomenon.

If not interfered with, collections of pus in the pleural space may become encysted, as previously mentioned, or may rupture into the lung with the evacuation of varying amounts of pus through the bronchi. The expectoration of purulent material may continue over a long period of time without the pleura being suspected as the source of the supply. This is more likely to be the case when the empyema is loculated, and not very large. In certain cases, with abscesses near the surface of the lung, it may be impossible at autopsy to determine whether one is dealing with an abscess which has ruptured into the pleura, or with an empyema which has perforated the lung.

More rarely, the purulent collection may burrow through the chest wall with the formation of a fluctuating abscess beneath the skin, in the event of which thoracic aneurism must be differentiated. The lower anterior surface of the chest is the region where these perforations occur, as the chest wall is thinnest in this area. Cough or forced expiration usually produce an increased tension in the abscesses, which is perceptible on palpation, and occasionally on inspection. The abscess eventually may perforate the skin, leaving a discharging sinus. The perforation of the diaphragm may lead to the development of a grave form of peritonitis, this complication being of a less favorable prognosis than the two previously mentioned. Perforation of the stomach, intestines, esophagus, or kidney by collections of pleural pus also have been reported. Septic thrombosis resulting from empyema may give rise to collections of pus in other parts of the body, by the formation of purulent emboli and infarcts.

After recovery from empyema there is almost invariably diminished expansion on the affected side, and this defect usually persists for some time, or becomes permanent. The compression of the lung and thickening of the pleura so frequently resulting from purulent effusions require a considerable time to reach a condition which is even approximately normal. This is shown, not only by the diminished expansion, but by flatness at the base; over the lower, duller portion the

breath- and voice- sounds are absent, and immediately above it lies a zone of bronchial breathing affording numerous fine moist râles. In time the lung tends to re-expand and the evidences of thickened pleura to disappear, change being more likely in the cases which have developed rapidly and have been operated upon early. The empyemata which have been allowed to persist for some time before being drained usually are accompanied by such an extensive connective tissue-formation in the pleura, and even in the lung, that complete re-expansion never takes place, and frequently is followed by contraction of the chest on the affected side and even by deformity of the spine.

### TREATMENT.

The dividing-line between serofibrinous and purulent effusions cannot always be closely drawn, even when exploratory puncture has been made, and the fluid itself studied. The general symptoms and conditions of the patient, the presence of a rational cause for the effusion, and the amount of fluid present will all have to be considered in deciding upon the treatment to be instituted. When the fluid is distinctly cloudy, and yet not of a decided purulent appearance, if a study of the cellular elements and bacteria present has not given evidence of sufficient weight to guide the further treatment, it may be sufficient to aspirate, with the understanding that an operation may be necessary upon the appearance of any reaccumulation, or if the patient's general condition fails to improve. When the fluid is evidently pus, operative interference should be insisted upon at once, for the evacuation of pus before the formation of a dense inflammatory exudate insures a better coaptation of the pleural surface, and may avoid the possibility of a long-continued, discharging sinus. The extension of the collection of pus into the lung, pericardium, through the chest wall or diaphragm, or the development of a general septicemia are dangers which also may be avoided by early operation.

While there are no physical signs which can be depended upon for determination of the presence of pus, there are certain findings suggesting that the pleural effusion is purulent and not serofibrinous; thus an acute onset with suddenly

developing symptoms, edema of the chest wall, high temperature, and evidence of toxemia, call for an immediate exploratory puncture. Hyperleucocytosis also suggests the probability of pus, although practically this laboratory finding seldom is of value on account of other coincident pathologic conditions capable of causing identical blood-changes.

Exploratory puncture should always be performed with care, especially in cases of long standing, or when the empyema results from, or is a complication of, pulmonary abscess, bronchiectasis, gangrene, interstitial pneumonia, or suppurative conditions in the lung. Perforation of the diaphragm, hemorrhage resulting from perforation of a blood-vessel in the walls of a pulmonary cavity, or bleeding from granulation tissue may lead to serious, if not fatal, results. The determination of the position of the diaphragm should be previously decided by means of a fluoroscopic examination in any case in which its location is doubtful.

The cases in which the greatest care is demanded are naturally those in which the collection of pus is small and walled off by adhesions from the general pleural cavity. These loculated empyemata may be situated between the lung and the chest wall, between the lung and the diaphragm, or in the spaces between the lobes. When occurring in the last two regions, their diagnosis may be attended by considerable difficulty.

The history and symptoms may suggest the presence of pus, and on examination localized tenderness, dullness, absent breath- and voice- sounds surrounded by a zone of fine râles, bronchial-breathing, and increased voice-sounds may be found. The *x*-rays may be of considerable value in confirming the physical signs and in enabling one more accurately to localize the collection of pus and the position of the diaphragm and heart. When the evidence points to a loculated empyema, an exploratory operation is to be preferred to exploratory puncture, on account of the difficulties attending the exact localization of the pus. The operation should be attempted only by an experienced surgeon, as it is not entirely devoid of danger, although not to the same extent as formerly, owing to improvement in the operative methods applied to surgery of the chest.

The operative treatment of empyema is distinctly a surgical question, the most important object being the establishment of free, constant drainage. To secure this usually it is necessary to remove a portion of several ribs, and the drainage tube should be constructed or arranged so as to prevent the admission of air as much as possible.

In the after-treatment of these cases the building up of the general health and strength of the patient is of the greatest importance. The re-expansion of the lung and the obliteration of the pleural space previously occupied by the collection of pus may be materially assisted by respiratory exercises when no concomitant disease of the lung forbids this measure.

### HEMORRHAGIC PLEURITIS.

The presence of blood in a pleural effusion is most frequently found in the secondary forms, although it may occur even in those of primary type. The most frequent cause is tuberculosis, although it is not uncommon in those due to malignant disease of the lungs and pleura. The effusions due to pneumonia may be blood-tinged, also those due to severe infections, or occurring during the course of chronic asthenic diseases or in purpura hemorrhagica. They possess very little diagnostic value, and their occurrence calls for no special line of treatment other than the measures ordinarily applied to effusions in general. Even the blood-tinged hydrothorax which occasionally occurs during the course of the cardiac or renal disease has no special significance, and calls for no particular remedial measures.

### PLEURAL NEOPLASMS.

The occurrence of primary carcinoma and sarcoma of the pleura is extremely rare, especially the latter, the disease usually arising secondarily to malignant disease in some other part of the body. In the early stages there is no feature to distinguish it from pleurisy of any other type, although usually it closely resembles that due to tuberculosis. The symptoms are not characteristic, consisting of pain on deep inspiration, with possibly some cough and dyspnea. With the



accumulation of fluid the dyspnea may become marked, and in the later stages the progressive loss and strength may suggest the presence of malignant disease. Fluid is almost invariably present, and presents no signs at first to indicate the process responsible for its accumulation. The diagnosis of malignant disease of the pleura is mainly reached by a process of exclusion, although occasionally signs which suggest its presence are evident. These signs depend upon the presence of thickening of the pleura due to malignant changes. Any effusion which tends to accumulate after repeated aspirations, and in which there is no evident cause for the presence of fluid, should be carefully observed as a possible case of malignant disease. This is especially true when the removal of the fluid is not followed by a decrease of the flatness and an increase in the amount of expansion on the affected side. When the impairment and other signs of thickened pleura persist after the removal of the fluid the probability of malignant disease is very strong, particularly should the chest fail to expand, and when there is progressive shrinking of the affected side. In some cases the resistance offered to the aspirating needle is of such a character as to indicate the presence of a thickened pleura, suggesting a tough, grating, fibrous tissue, quite different from the soft, smooth, inflammatory exudate of the non-malignant conditions. In some cases small nodular growths may arise at the point where the needle has been inserted on some previous occasion for the withdrawal of the fluid, from an extension of the pleural process along the path of the needle. When these nodules are found they should be excised and examined microscopically, as their gross appearance resembles very closely that of the nodules which may arise along the needle-path in tuberculous effusions. The examination of the fluid occasionally may give information of value in arriving at a correct diagnosis (see *Pleural Fluids*, p. 499). A careful microscopic study of sections made of the small plug of tissue which occasionally occludes the aspirating needle, has in some cases provided information upon which a positive diagnosis of malignancy could be based.

The *treatment* of a pleural malignant neoplasm is extremely unsatisfactory, for it consists entirely of carrying out of such measures as may relieve the symptoms temporarily. Removal

of the fluid at frequent intervals is usually conducive to the comfort of the patient by relieving the cough and dyspnea. In nearly every case opium and its derivatives have to be persistently employed to give the patient relief from the pain and distress which eventually supervene in practically every case. In some patients the removal of the fluid appears to aggravate the symptoms, rather than to provide relief, and in these only such an amount of fluid should be withdrawn as may be necessary to relieve grave pressure-symptoms. In the average case usually it is satisfactory to tap the chest at fairly frequent intervals to prevent the accumulation of a large amount of fluid, the repeated removal of small quantities of fluid proving less distressing to the patient than the withdrawal of a large amount at single *séance*.

### HYDROTHORAX.

When the transudation of serous fluid into the pleural cavity is sufficient in amount to be recognized clinically, it is usually due either to cardiac or to renal disease, the treatment for the relief of this symptom depending upon the disease responsible for its development. When the fluid persists, in spite of the correction of the provocative disease, or when the respiratory or circulatory functions are seriously interfered with, the fluid should be aspirated, with extraordinary precautions against infections, shock, edema, etc., as stated in the section on Serofibrinous Pleuritis (see p. 517). The physical signs of a hydrothorax are the same as those of any other collection of fluid in the pleural space, to which may be added certain symptoms and signs incident to cardiorenal and cardiovascular diseases.

### HEMOTHORAX.

This name should be reserved for those cases in which there is an actual extravasation of blood into the pleural space, whether due to injury of the chest wall or lung, or to the rupture of a large vessel in the mediastinum or chest wall, as a result of erosion or aneurismal dilatation. The mere occurrence of a blood-tinged fluid in the pleural space should not be considered as a true hemothorax.

The *treatment* of this condition is purely surgical in the large majority of cases, especially as they are most commonly traumatic in origin. If the hemorrhage is not very copious, the consequent hemothorax may be completely absorbed, even when fairly large coagulation with subsequent absorption may take place; although as a rule the absorption is not complete, and a portion of the clot usually undergoes organization.

The principal danger in this condition is infection of the extravasated blood, which is particularly prone to occur when the condition is due to trauma, in perforating wounds of the chest wall or rupture of the lung.

The occurrence of hemorrhage into the pleural space calls for the general remedial measures applicable to hemorrhage in any other part of the body—surgical measures to secure the bleeding-point, and absolute rest, with immobilization of the affected lung by adhesive plaster straps to the chest. If alarming pressure-symptoms develop, thoracentesis may become necessary, or if infection of the effusion becomes evident one may have to resort to thoracotomy.

### CHYLOTHORAX.

An actual chylothorax, or chylous pleural effusion, due to pressure upon, or rupture of, the thoracic duct, should not be treated by aspiration until the level of the fluid has remained stationary for some time, or the pressure-symptoms have become so grave as to demand interference. When the effusion is merely chyloform, and due to fatty degenerative processes, or to the presence of some albuminoid substance, the treatment should be that of the ordinary effusion. The diagnosis of this form of pleural effusion can be made only by an examination of the fluid obtained by exploratory puncture or aspiration.

### PNEUMOTHORAX.

This condition consists in the presence of atmospheric air or gas within the pleural sac. The lung is an elastic organ held in apposition to the chest wall by the negative pressure within the pleural space. The entrance of atmospheric air into the pleural space results in collapse of the lung through

destruction of the intrapleural negative pressure, which maintains the surface of the lung in close apposition with the chest wall, permitting it to contract by reason of its normal elasticity. Pneumothorax should not be looked upon as a compression of the lung by air-pressure, but rather as a removal of the force which has maintained the contractile lung in a position of overdistension. As atmospheric air is comparatively rapidly absorbed from the pleural cavity, one may readily understand that the course of this process may vary with the exciting conditions, depending upon whether the opening which forms communication between the pleural space and the outer air through the lung or chest wall persists, is quickly closed, or is valvular in character.

Atmospheric air may gain entrance to the pleural space through the chest wall, as a result of penetrating wounds (gun-shot wounds, stab wounds, thoracotomy, thoracentesis, etc.); or from perforation of the pulmonary pleura and lung, trachea, bronchi, stomach, esophagus, or intestines, as a result of trauma or disease. Rarely, pneumothorax may result from the formation of gas by certain types of micro-organisms developing in pleural effusions, as cases have been reported in which gas-forming bacteria in pleural effusions have appeared to be responsible for the formation of the pneumothorax.

From a clinical standpoint, interest is chiefly centered in the cases due to perforation of the lung as a result of disease. By far the commonest cause of this type of pneumothorax is pulmonary tuberculosis, but it may occur rarely in pulmonary abscess, gangrene, bronchiectasis, infarction, malignant disease, empyema, or possibly emphysema. A few cases have been reported in which a pneumothorax developed from rupture of the lung in persons who were apparently in perfect health, or in whom there was no evidence of any disease capable of producing pneumothorax. Apparently here it arose as a result of some unusual violent exertion, such as coughing, sneezing, laughing, and in those exceptional instances where it developed while the subject was at rest, or even asleep, no tangible factor could be discovered.

The conditions arising after the entrance of air to the pleural space are dependent upon whether the pleura is free or partially adherent. When the pleura is free the lung col-

lapses completely, retracting toward the root of the lung, and at autopsy it may be found as a small shrunken mass lying against the spinal column. When adhesions are present, their location and extent regulate the degree of collapse, and the situation of the accumulation of air. Usually the collapsed lung is the seat of a distinct tuberculous process, in which case the perforation can be easily demonstrated, commonly in the wall of a superficial cavity. As the perforation in the lung is usually the result of disease, infective material frequently gains entrance to the pleural space, and results in hydropneumothorax, pyopneumothorax, or rarely h  mopneumothorax. It is exceedingly rare to find a pneumothorax which has existed for any length of time in which fluid is not present. The presence of air in the pleural space is usually accompanied by displacement of the heart, diaphragm, liver, spleen, and even the opposite lung.

The onset of the disease varies greatly, although, as a rule, it occurs suddenly with violent, sharp pain, severe dyspnea, and an intense air-hunger and sense of suffocation. In other patients there are merely slight pain and moderate dyspnea, or symptoms may be entirely absent, and the condition discovered only in the course of routine examination. The severity of the symptoms at the onset of the disease probably are largely dependent upon the degree of collapse of the lung as controlled by the extent of adhesions.

While small quantities of air in the pleural space may be difficult to detect, in the average case the signs are so characteristic and striking that the condition is easily recognized. On inspection usually there are evidences of dyspnea, immobility of the affected chest, distended, bulging interspaces, absence of the diaphragm shadow, and possibly displacement of the heart, as recognized by the abnormal position of the apex-beat. Vocal fremitus is absent, and displacement of the solid viscera may be determined by palpation. The percussion-sound is usually hyperresonant, loud, and low-pitched; although when the pulmonary opening is large and patulous the note may be tympanitic, or if the air in the pleura is under very great tension, as may occur when the opening is valvular, the sound may be dull. The area of hyperresonance may extend beyond the median line, encroaching upon the normal



side. Percussion also may be employed to confirm the displacement of the heart, or suggest the presence of fluid by the area of flatness at the base. The breath-sounds are decidedly suppressed or absent, although occasionally amphoric breathing is audible together with râles of a distant, amphoric quality. The most striking and characteristic phenomena are the metallic tinkle, the succussion splash, and the coin-test. In the early cases without fluid usually the first suggestive group of signs is absence of breath- and voice- sounds, distension and immobility of the chest, and hyperresonance on percussion. The *x*-rays are of considerable value in determining the presence of small collections of air, and for confirming the diagnosis of pneumothorax in doubtful cases. The presence and amount of fluid in these cases may also be conveniently studied by the *x*-rays. In the average case radiology is unnecessary, the physical signs alone being usually sufficient to determine the size and location of the pneumothorax and the quantity of fluid.

#### TREATMENT.

The treatment of pneumothorax depends upon the cause of the condition, the presence or absence of disease in the lung, and the amount and character of the effusion which so commonly accompanies the process. The medical care of pneumothorax may be conveniently described under two headings, one for the relief of symptoms, and the other such measures as may be directed toward assuring the closure of the opening through which the air gains entrance to the pleura.

When due to the entrance of atmospheric air from an opening in the chest wall or through a healthy lung, the treatment consists chiefly of relieving symptoms, securing absolute rest of the lung, and applying measures designed to prevent infection and hemorrhage. The large majority of such cases tend to heal spontaneously when merely kept at rest. Rest in bed, strapping of the chest, relieving grave pressure-symptoms by release of a moderate amount of the air, and sufficient morphin to relieve pain and to control distressing cough, is usually sufficient. In removing by thoracentesis the air enclosed in the pleural space as a result of rupture of the lung, care must be exercised not to exhaust the air completely,

since a certain amount of pressure is necessary to maintain a closure of the pleuro-pulmonary fistula. In those cases in which the condition develops very rapidly; with severe dyspnea, cyanosis, and evidence of cardiac failure or suffocation, the removal of a certain amount of air by thoracentesis is necessary in order to save life. When wounds of the chest are the factor, such steps must be taken as will check any tendency to bleeding, and cleanse the wound of infective material. When atmospheric air has gained entrance to the pleura through a non-patulous opening that does not leak continuously, the intrapleural air tends rapidly to become absorbed. It is only when additional quantities of air are constantly being added to that already within the pleura that a pneumothorax persists.

The most frequent cause of pneumothorax is pulmonary tuberculosis, in which a small subpleural cavity or caseating area ruptures into the pleura and leaves a patulous opening, through which air gains entrance to the pleural space; or the opening may be valvular, in which case air may readily gain entrance to the pleura, but is prevented from returning. The usual location of the air collection is not at the apex, as one would expect, but over the lower portion of the lung, as the upper part of the lung is usually adherent to the chest wall. In these cases not only air, but infective material from the diseased lung, gains entrance to the pleural space, so that the pneumothorax usually is complicated by a serous or purulent effusion. A moderate amount of serous effusion should never be disturbed in these cases, as it not infrequently aids in securing closure of the opening in the lung. Even when the effusion becomes massive, great care must be used in withdrawing the fluid; in such instances it is best to aspirate sufficient merely to relieve pressure-symptoms, for the entire effusion should never be withdrawn so long as there is a possibility of reopening the pulmonary fistula.

When pyopneumothorax develops, however, the presence of a large quantity of pus of itself constitutes such a serious menace to life and health that its removal is imperative, either by thoracentesis or by open operation and drainage, according to character of the pus and the evidence of absorption of toxic material by the patient. When the quantity of pus is

small, and there are no signs pointing toward a general toxemia, it may be allowed to remain undisturbed until sufficient time has elapsed to permit a closure of the opening in the lung.

It has been recommended that in the cases of pulmonary tuberculosis complicated by pneumothorax, when serofibrinous or purulent effusions develop, their removal may be accomplished even before the opening in the lung has had a chance to close by replacing the fluid with nitrogen gas. The fluid is withdrawn and nitrogen gas introduced simultaneously, maintaining by this means a constant pressure within the pleural space, this pressure being controlled during the exchange by the use of the water manometer. This procedure has many points to recommend it, not the least of which is the advantage obtained by the compression of the lung upon the tuberculous process. A sufficient number of cases favorably treated by this method have been reported to warrant its being more generally adopted (see Tuberculosis-Pneumothorax, p. 462).

### **BACTERINS IN THE TREATMENT OF DISEASES OF THE LUNGS, BRONCHI, AND PLEURA.**

Throughout the text repeated reference has been made to the use of bacterial vaccines or bacterins in certain diseases of the bronchopulmonary system, and it seems advisable to consider the subject a little more fully in a special section, on account of the rather general adoption of this method of treatment. The subject is one which one hesitates to discuss, as it is difficult to estimate its value, and in view of the many reported cases in which it is difficult to determine how much could be credited to the employment of bacterins and how much to other measures. The administration of bacterins in self-limited diseases, or those in which abortive types are frequently encountered, is another possible source of error. The writer does not believe that it has been absolutely demonstrated beyond question that the injection of bacterins is absolutely without danger in many of the bronchopulmonary diseases in the treatment of which they have been recommended.

In the first place the most serious difficulty lies in securing a culture of the one or several micro-organisms responsible for

the disease. The various methods for washing the sputum in repeated changes of water, preceded by careful cleansing of the mouth and pharynx, have added considerably to our ability to obtain secretion from the bronchial tract with little or no contamination. Having obtained a specimen of bronchial secretion, the problem still remains of determining which bacterium or group of bacteria is responsible for the process. The discovery of micro-organisms in pure culture under such conditions may be said practically never to occur. The custom has been to make up a mixture of the various recognized pathogenic micro-organisms isolated, and to prepare a final bacterin containing a definite proportion of each, so that the dose of each germ may be accurately estimated. By this method the relative number of each type of micro-organism is definitely fixed, inasmuch as it is impossible to vary the proportion of any one type or to omit any if it should seem desirable. The preparation of bacterins from each single type of bacterium seems to obviate this objection, but it makes their administration much less convenient. It has been recommended that a careful study be repeatedly made during a course of treatment by mixed bacterins, so that the administration of certain micro-organisms might be discontinued as they were found to have disappeared from the sputum. The question of treatment by means of bacterins would be very much simplified if one could be certain that the predominating micro-organism was the most important etiologic factor, but apparently this is not the case. Further confusion is caused by the fact that certain micro-organisms require the presence of other, apparently innocuous, bacteria before they are capable of setting up a morbid process.

The list of germs capable of causing infection of the respiratory tract is gradually increasing in numbers; seemingly among the most important are the pneumococcus, streptococcus, influenza bacillus, bacillus septus, micrococcus catarrhalis, staphylococcus, micrococcus tetragenes, pyocyaneus bacilli, bacillus coli, bacterium aërogenes, mucosus group, and the typhoid bacillus. The relative frequency with which they are found in diseases of the respiratory tract varies from year to year. The futility of employing stock bacterins in the treatment of any disease of the respiratory tract must be per-



fectly obvious, unless a thorough study of the case should happen to reveal a condition of affairs in which the stock vaccine is peculiarly and specially applicable.

The most that can be expected from the use of vaccines in acute conditions is a limitation of the disease, and in any infectious condition their only possible utility is in increasing resistance to the invading micro-organisms, which in many diseases of the respiratory tract is only one of several conditions which call for treatment. In recent years we have become so accustomed to view disease from the standpoint of the infecting micro-organisms alone, that the response to or effect upon the tissue of the host has been somewhat ignored. To cite an example, in acute bronchitis the mere overcoming of the invading bacteria is in itself not sufficient to restore the function of the bronchi in many instances, and the cases usually require further treatment to establish a cure. In no case should vaccines be employed to the exclusion of the regular methods of treatment.

In the employment of bacterins for therapeutic purposes there are certain cases in which one must proceed cautiously. This is true of persons who give a history of sensitization to foreign proteins, whether shown by severe reaction to variola vaccination, susceptibility to the presence of horses or small rodents, or cutaneous reactions to various foodstuffs. In patients in whom there is marked cardiac or renal disease, or those who are rapidly progressing toward a fatal termination, with marked asthenia, bacterin treatment should never be employed. Any acute conditions of the kidneys, as evidenced by blood, casts, and albumin in the urine, constitutes a distinct contraindication to their use. In diseases of the respiratory tract the administration of bacterins is especially likely to be followed by an increase of symptoms and signs, which may prove a very serious matter in such conditions as capillary bronchitis, or the bronchopneumonia of children. Patients being treated by bacterins should be warned of the probability of an aggravation of symptoms after the first injection.

Space will not permit of describing in detail the various micro-organisms, dosage, etc., to be employed in every disease of the respiratory tract, but there are a few general rules



and precautions which may be given. Great care should be exercised in the preparation of the bacterin to see that the numerical proportion of bacteria to the definite amount of solution be accurately ascertained, so that the dosage may be exactly gauged. One must be positive that the bacterin is absolutely sterile—that the micro-organisms contained therein are not viable. The original suspension of the bacteria should be made as concentrated as possible, dilutions being made from this stock solution at the time of the injection. Unless the suspension is concentrated, disintegration of the bacteria takes place rapidly. The bacterin should be prepared so that the number of bacteria may be estimated on the basis of a mil or a decimal thereof, as most of the syringes used for the injection are scaled according to the metric system.

The injections should be made in the loose subcutaneous tissue at a point where there is no pressure from the clothing, the arm and infraclavicular regions being very convenient sites for the operation. The injected material should be well spread out under the skin, unless there is fear that the reaction will be pronounced. Under this circumstance the material should be allowed to remain at one point so that absorption will be slow; with this technic the local reaction may thus be more marked, but the general reaction will be milder. The injections should be given before three o'clock in the afternoon, so that slight reactions in the first eight hours may be observed.

The dosage varies in different conditions and with various micro-organisms, but a few general rules may be given as follows: When the extent of the lesions is great the individual doses should be small, and large doses should be given when the focus of disease is small. When the part affected has a rich blood-supply small doses are indicated, and when it has a poor blood-supply large doses should be used. No reinjection should be given while there is any evidence of the previous injection. The dose should never be increased so long as the condition is improving. The first and second doses should never be given during the menstrual period. The dose should be sufficient to produce a mild reaction, as evidenced by an increase of secretion from the mucous membrane of the part affected, and severe reactions should be avoided. The evidence of reaction may be taken to determine the intervals

between doses, reinjection never to be made sooner than twenty-four hours or later than seventy-two hours after all the effects of the previous injection shall have disappeared. A moderate increase in the number of leucocytes in the circulating blood at the end of twenty-four hours has a favorable significance, but if the count of these cells diminishes the succeeding dose should be less than the one preceding.

In conclusion it may be stated that the results from bacterin treatment are frequently disappointing, occasionally striking, and in some instances unfavorable. The cases which are most resistant to the ordinary methods of treatment, such as bronchiectasis and the chronic bronchitis of emphysema, in which much good was anticipated from the use of bacterins, unfortunately constitutes a group in which they seem to be of the least value. Probably the use of bacterins eventually will be found to be of value for the prevention of acute diseases of the respiratory tract, rather than for therapeutic purposes. Before this hope is placed upon a firm basis, there will have to be many careful studies of the bacteriology of these processes, an improvement in our methods of determining the causative micro-organisms in the individual case, and possibly a more accurate method evolved for the determination of the proper mode of application, so far as the dosage and frequency of administration are concerned.

The writer begs to acknowledge his indebtedness to Dr. Herbert Fox for much of the material upon which the foregoing section is based, at the same time expressing his regrets that he finds himself unable (possibly on account of a more limited experience with this method of treatment) to share with Dr. Fox his optimistic views in regard to the value of bacterin treatment.

# Diseases of the Kidneys

BY

JUDSON DALAND, M.D.,

Professor of Clinical Medicine, Graduate School of Medicine, University of Pennsylvania,

AND

FRANCIS J. DEVER, M.D.,

Formerly Associate Professor of Clinical Medicine, Medico-Chirurgical College; Assistant Visiting Physician, Philadelphia General and Medico-Chirurgical Hospitals.



# Diseases of the Kidneys.

---

## FOREWORD.

PRECEDING the discussion of diseases of the kidneys, a small amount of space is devoted to the essential features of the gross and minute anatomy of these organs. A lengthy discussion of the various theories of the mechanism of the urinary secretion is avoided; but a brief summary of the modern views of renal physiology is presented. The normal urine is described, and simple tests are given for the detection of the various normal constituents.

A classification of movable kidney is made based upon the degree of mobility. A word of warning is given against attaching undue importance to the accidental discovery of movable kidney. Operation is believed to be contraindicated, except under a few well defined circumstances. Treatment along the lines of general upbuilding, with exercises to increase the tone of the abdominal muscles, is described. The wearing of a belt designed to keep the kidney in place is viewed as a temporary measure, to be employed only until the abdominal muscles can be developed by appropriate exercises and the emaciated fattened.

It is rarely possible, clinically, accurately to differentiate the different types of nephritis described by the pathologist. Indeed, the pathologist is unable at times to decide to which type a particular pair of kidneys belong when he has them in his hands for examination. Therefore it is deemed sufficient, from the standpoint of treatment, to classify nephritis as: (*a*) acute parenchymatous or acute diffuse nephritis; (*b*) chronic parenchymatous or diffuse nephritis without induration; and (*c*) chronic interstitial or chronic diffuse nephritis with induration. The arteriosclerotic kidney is considered with chronic interstitial nephritis.

The importance of focal sepsis as a cause of acute nephritis, and ultimately chronic nephritis through frequent recurrences,



is emphasized. Much information of value in prognosis and treatment is derived from a study of the function of the kidney in disease. No one test alone is capable of throwing sufficient light upon the function of the kidney as a whole. It is therefore recommended that the ability of the kidney to excrete the dye, phenolsulphonephthalein, and also the degree of retention in the blood of uric acid, urea, and creatinin, be studied. A technic for these tests is given, and the use of a test diet, according to the method described by Mosenthal, is described. Tests for albumin, blood, etc., in the urine are given, and the significance of casts and cylindroids is discussed.

No mention of certain drugs and combinations, still in great vogue, is made under the heading of treatment of nephritis, because the authors believe them to be without value. Certain other drugs are condemned because their use is likely to irritate still further the diseased kidneys. Diet and elimination through the skin occupy an important place in the treatment, and the technic of the various measures employed by the authors is described. The treatment of the various clinical types of Bright's disease is discussed in detail.

In addition, consideration is given to renal lithiasis, pyogenic infections, hydronephrosis, and renal tuberculosis.

### GENERAL CONSIDERATIONS.

Briefly to rehearse the chief features of the *clinical anatomy* of the kidneys, it will be recalled that they lie behind the peritoneum in a mass of fat and loose areolar tissue, alongside the vertebral column, opposite the twelfth thoracic, and the first, second and third lumbar vertebræ. They are approximately  $4\frac{1}{2}$  inches (11.5 cm.) long, 2 to  $2\frac{1}{2}$  inches (5.08 to 6.3 cm.) broad, and  $1\frac{1}{2}$  inches (3.8 cm.) thick, and weigh  $4\frac{1}{2}$  to 6 ounces (139.9 to 186.6 Gm.). The kidneys of females weigh a trifle less. Immediately above the kidneys lie the suprarenal glands.

The anterior surface of the right kidney is in relationship with the inferior surface of the liver, the second part of the duodenum, small intestine and the hepatic flexure of the colon. The part of the kidney in relation with the liver and intestine is covered by peritoneum, while that portion in

relation with the duodenum and colon is devoid of a peritoneal investiture.

The anterior surface of the left kidney is in relation with spleen, stomach, pancreas, small intestine, and splenic flexure of the colon. The area in contact with the stomach is covered by peritoneum of the lesser sac; that in relation to the small intestine by the peritoneum of the greater sac.

The posterior surface, entirely devoid of peritoneal covering, lies upon the diaphragm, the lumbar aponeurosis, the arcuate ligaments, the psoas and transversalis muscles, the last thoracic, iliohypogastric, and ilioinguinal nerves. The diaphragm separates the kidney and pleura, but very commonly the diaphragmatic fibers are defective or absent, so that the perirenal alveolar tissue is in contact with the diaphragmatic pleura.

In the funnel-shaped cavity of the renal pelvis is the ureter which, passing over the psoas muscle, converges toward the ureter of the opposite kidney and passes obliquely through the wall of the bladder. It is a musculo-membranous tube, the upper end of which is expanded, and within the renal pelvis, divides into a number of short tubes called calyces, each of which embraces the apex of a Malpighian pyramid. The capacity of the renal pelvis is from 5 to 15 mls (1.3 to 4.0 f3).

The kidneys are held in position by the apposition of the neighboring viscera and the *fascia renalis*, which blends with the fascia on the quadratus lumborum and psoas muscles, and thus is attached to the vertebral column. Above, the fascia blends with the fascia of the diaphragm. A smooth firm covering of fibrous connective tissue forms the renal capsule, which strips easily, leaving a smooth, even surface, deep red in color. The capsule enters the hilum of the kidney, blending with the connective tissue carried in with the vessels and nerves.

The renal substance is divisible into an outer portion, the *cortex*, which is  $\frac{1}{3}$  to  $\frac{1}{2}$  an inch (8.4 to 12.7 mm.) in thickness; and an inner portion, the *medulla*, which is  $\frac{2}{3}$  to  $\frac{3}{4}$  of an inch (16.9 to 19.05 mm.) thick.

The cortex lies immediately beneath the capsule, and sends projections toward the sinus, between the pyramids,

forming the columns of Bertini. It is made up of the convoluted and straight tubules, Malpighian bodies, blood vessels, nerves, lymphatic and connective tissue. The straight tubules are lighter colored than the rest of the cortical structure, and are visible as ray-like prolongations (medullary rays) extending toward the external surface of the cortex.

The medulla is made up of the pyramids of Malpighi, of which there are from eight to twenty. They are composed of the straight collecting tubules, and part of the ascending and descending limbs and loops of Henle.

For their *blood supply* the kidneys depend upon the renal arteries, which are given off from the abdominal aorta. Just before entering the kidneys through the hilum, each artery divides into four or five branches which enter the substance of the organ between the pyramids. At the juncture of the cortex and medulla the arteries run a course about parallel with the surface of the kidney, giving off branches to the cortex and medulla. The branches to the cortex send off twigs to each Malpighian corpuscle.

The nerve supply is through the renal plexus, which is formed by branches from the solar plexus, the lower and outer part of the semilunar ganglion and aortic plexus, and from the lesser splanchnic nerves. The nerves of the kidney communicate with the spermatic plexus. The spinal segments corresponding to the kidney and the ureter are the tenth, eleventh and twelfth dorsal and first lumbar.

Little or no motor force is supplied by the nerves to the renal pelvis. Sensation probably is not very acute, as is demonstrated by the slight response to the presence of the tip of a catheter, or slight distension of its walls by fluid. When the stimulation is sufficiently great the response is obtained not only from the renal pelvis, but from the entire urinary tract as well.

The arteries which form the Malpighian corpuscles in the cortex end in a tuft of convoluted capillaries, which are surrounded, or, more correctly speaking, invaginated, by a hyaline membrane called the capsule of Bowman. From this capsule is given off a narrow tubule which soon becomes wider and convoluted, and is called the proximal or first convoluted tubule. Approaching the medulla they become

less convoluted, then spiral, and enter the pyramids as straight tubes forming the descending limb of the loop of Henle. Descending toward the apex of the pyramid, they suddenly turn, making the loop of Henle, and ascend as a straight tubule (ascending limb) to the cortex, where they again widen and become convoluted (distal or second convoluted tubules), and arch into the straight collecting tube which passes from the cortex to the medulla, receiving in its course through the cortex a number of arches from other distal convoluted tubules. Toward the apex of the pyramid several straight collecting tubules unite to form a large tubule, finally ending in the papillary or excretory duct at the apex of the pyramid. The tubules consist throughout of a single layer of cells on a basement membrane.

The character of the cells varies in different parts of the tube. The capsule of Bowman and the adjoining narrow tubule are lined with squamous cells. The first or proximal convoluted, the spiral tubules, and the second or distal convoluted tubules are lined with irregular columnar cells; the descending limb of Henle's loop is lined with simple squamous cells; the loop, the ascending limb and the arched collecting tubes are lined with cuboidal cells. The straight collecting tubules are lined with columnar cells.

The kidneys are an important unit of the excretory system of the body, the other units being the skin, lungs, liver and intestines. There is a close interaction among these units, and under certain conditions and in a variable degree they compensate each other. This compensatory action is frequently observed clinically between the skin and the kidneys, and forms the basis of treatment in those diseases of the kidney characterized by functional insufficiency.

The excretory product of the kidneys is the urine. The exact manner of its elaboration is still unknown, but the latter-day theories may be summarized by regarding the water and its salts as a product of filtration through the glomeruli; the dissolved components, as urea, uric acid, etc., are the products of the activity of the cells lining the uriniferous tubules.

The filtration of water through the glomeruli is in direct relation to the pressure in the renal artery. A high pressure

is maintained in the glomeruli, due to the fact that the vessels entering them are of greater diameter than those of exit. Filtration, however, is not the sole function of the glomeruli, as their epithelium has a selective action in removing some of the salts and, at the same time, preventing the passage of the serum albumen of the blood.

Clinically, the theory of the dual mechanism of filtration and secretion by the kidneys is supported by the very common observation that in certain types of renal disease evidence of a chronic toxemia (uremia) is present, even though the daily amount of water eliminated is normal or even increased; the probable explanation being that the ability of the kidneys to filter the water from the blood is normal, but the cells are unable to eliminate the toxins. Under such conditions the urine is pale in color and of low specific gravity. That the nervous mechanism of the kidneys plays a rôle in the secretion of urine is certain, but to what extent is unknown. As yet no true excretory nerve has been discovered, but the vasodilator and vasoconstrictor functions have been observed and studied by physiologists. Experiments have demonstrated that stimulation of the vasodilator nerves increases the flow of urine, while stimulation of the vasoconstrictors has an opposite effect. The frequent and small urination under the influence of mental apprehension is well known to the layman. Polyuria in hysterical individuals, with normal kidneys, is a common occurrence.

Fresh *normal urine* is a clear liquid with a specific gravity of 1015 to 1020. The color varies from a pale yellow to a reddish brown, and is due to the presence of urobilin, urochrome and uroerythrin, all of which are derived from bile pigments. The normal reaction is acid, due to the presence of acid phosphates of calcium and sodium. When neutral and alkaline, unless from decomposition before or after leaving the body, the cause is usually some metabolic disturbance, or, as in cystitis, a disease. Upon voiding, the temperature is approximately 100° F. (37.7° C.). When, after standing, it cools to room temperature, a light cloud is observed, composed of mucus and epithelial cells. Frequently, when patients observe this cloud or turbidity from phosphates they



become frightened and believe that they have a serious disease of the kidneys.

The average normal quantity of urine excreted in twenty-four hours is 50 ounces (1500 mils), or, roughly speaking, 1 ounce (30 mils) per kidney per hour. The amount varies with the weight of the patient, the amount of liquid ingested, and the amount of liquid lost through the other organs of excretion, *i.e.*, lungs, skin, and the gastrointestinal tract. For example, profuse sweating or diarrhea will cause a reduction in the amount of urine, and the imbibing of a large quantity of liquid or the vasodilation of the renal capillaries, secondary to the constriction of vessels in the skin (*e.g.*, effect of low temperature) will increase the amount of urine. Reference has already been made to the influence of the nervous system, through the vasomotor apparatus.

It is estimated that in 1000 mils (33.8 f $\bar{5}$ ) of urine, solids are present to the extent of forty parts, composed of urea, uric acid, hippuric acid, creatinin, ammonia and various inorganic salts, such as chlorides and sulphates of various metals. The total urinary solids may be estimated approximately by multiplying the last two figures of the specific gravity by the coefficient of Haeser, 2.33. The result expresses the total solids in grams per 1000 mils (33.8 f $\bar{5}$ ).

Another method is that of Metz, by which the last two figures of the specific gravity are multiplied by 0.00233, and this product by the total number of cubic centimeters of urine excreted in the twenty-four hours. The final product will be the total weight of solids expressed in grams. Suppose the quantity of urine excreted in the twenty-four hours is 1500 mils (50 f $\bar{5}$ ), with a specific gravity of 1020. Then  $20 \times 0.00233 \times 1500$  mils (50 f $\bar{5}$ ) = 69.9 grams (1083.4 grs.) of solids in that particular day's output.

Of the solids, urea (CON<sub>2</sub>H<sub>4</sub>) is the most abundant and, perhaps, the most important. The amount excreted each day varies from 463 to 617 grains (30 to 40 Gms.), representing an amount of metabolized protein equivalent to 3 to 4 ounces (90 to 120 Gms.) derived from the protein of the tissues of the body and that of the food ingested. The urea derived from the tissue protein is a fairly constant factor, while that derived from the protein of the food is variable, as shown by the fact

that the amount of urea varies with the amount of protein consumed. Just what particular body tissue or tissues undergo the protein metabolism is not known with certainty. The absence of a parallelism between urea production and muscle work leads to the conclusion that there is but little protein metabolism in muscle tissue.

The liver is probably the chief seat of urea formation. It is noteworthy that in diseases of the liver characterized by destruction of the organ, *e.g.*, acute yellow atrophy, abscess, etc., the amount of urea excreted is diminished, while the amount of ammonium salts in the urine is increased. Experimentally, when the portal vein is anastomosed with the ascending vena cava, thus excluding almost completely the action of the liver on the products absorbed from the intestinal canal, the output of urea is diminished and the ammonium in the urine is increased. The antecedents of urea, *i.e.*, those products from the intestinal canal from which urea is directly manufactured in the liver, are the carbonate, carbamate and lactate of ammonia. Two molecules of water are abstracted by the liver cells from these products, forming urea, as shown in this formula:



The ammonia is derived from the proteins by hydrolysis and cleavage. The amino-acids, as tyrosin, leucin, glutamic and aspartic acids, diamino-acids and bases, as lysin, arginin, histidin, which are also products of the hydrolysis of proteins during digestion, are capable of being absorbed as such by the epithelial cells of the villi and mucous membrane, in which they undergo a cleavage into an  $\text{NH}_2$  portion and an organic portion; the former is then converted into ammonia and subsequently into urea by the liver cells. The intestine is not the sole source of the ammonia and the amino-acids. There is evidence that the proteins that enter into the composition of all tissues and tissue fluids are undergoing at all times a hydrolysis, under the influence of enzymes, whereby products are produced similar to, if not identical with, those produced in the intestine. These, after their discharge into the bloodstream, are carried to the liver, where they undergo the same change as though derived from the intestine. A certain

amount of urea arises from the further metabolism of uric acid. It is estimated that about one-half the uric acid formed in man is converted into urea.

The best and simplest method for the quantitative estimation of urea for clinical purposes is Knop-Hüfner's method, which depends upon the decomposition of urea into carbon dioxid, water and nitrogen, by means of a solution of sodium hypobromite in an excess of sodium hydroxid. The nitrogen liberated from a definite amount of urine is measured volumetrically, and from this the corresponding amount of urea is calculated. The carbon dioxid is absorbed by the excess of sodium hydroxid. The sodium hypobromite solution may be prepared as follows: Seventy mils of a 30 per cent. sodium hydroxid solution are mixed with 180 mils (6 ozs.) of water and 5 mils of bromin. The latter dissolves with the formation of sodium bromid and sodium hypobromite.



As soon as the bromin has dissolved, the reagent is ready for use. It should be freshly prepared every day, and should never be used warm. A very convenient and economical way is to have the reagent made up as two solutions. Solution A is a 25 per cent. sodium hydrate; Solution B consists of bromin 1 part, potassium or sodium bromid 1 part, water 8 parts. For the test, 1 part of bromin solution is added to 20 parts of the sodium hydrate solution. The Doremus or the Doremus-Hinds ureometer is used.

The Doremus ureometer consists of a bulb with an upright graduated tube and a pipette graduated to hold 1 mil of urine. The graduation on the upright ureometer reads in grams of urea per cubic centimeter of urine. When a determination is to be made the upright tube and about half of the bulb are filled with the hypobromite solution. Care must be taken to exclude all air from the tube. By means of the pipette 1 mil of urine is introduced into the upright tube and a sufficient time allowed for the evolution of gas to cease, usually about ten to fifteen minutes, at the end of which time the lower meniscus of the solution is read off upon the scale. This reading, multiplied by the number of cubic milliliters in the twenty-four-hour specimen, will give the quantity of urea

excreted in twenty-four hours expressed in grams. In studying the urea excretion it is necessary to estimate the quantity in a twenty-four-hour collection of urine, and a record of the quantity and kinds of food eaten should be kept during the collection of the specimen.

**Uric Acid** ( $C_5H_4N_4O_3$ ) is a normal constituent of the urine, varying in quantity from 2 to 15 grains (0.13 to 0.9 Gm.) in twenty-four hours. It probably does not exist in a free state in the urine, but is combined with sodium and potassium in the form of a quadriurate. The urates, when in excess, are frequently deposited from the urine as a brick-red sediment, the color being due to their combination with the coloring matter uroerythrin. When pure, uric acid crystallizes in the rhombic form, though it may assume a variety of other forms. It is a cleavage product. Nuclein, a constituent of the nuclei of all cells, yields nucleic acid during metabolism, and from the latter uric acid is derived. Nucleic acid, when decomposed, yields a series of bases, such as xanthin, hypoxanthin, adenin, guanin, etc., which, because of the fact that they can also be obtained from a synthesized body called purin, are called collectively the purin bases. There is a close connection between these bases and uric acid. It is likely that uric acid is derived from one of them, probably hypoxanthin. Uric acid in the body has an endogenous and exogenous origin, *i.e.*, it is derived from the nuclein of the body cells and from the cells of ingested animal food. In a disease characterized by a very great increase in the number of leucocytes, as in leukemia, uric acid excretion is increased.

*The Murexid Test.* Add a drop of nitric acid to a small quantity of urine in a porcelain dish and evaporate to dryness. After cooling, allow a drop or two of ammonia water to come in contact with the residue, and in the presence of uric acid or urates, a bright blue-violet color will be produced.

*Schiff's Test.* Add a drop of nitric acid to a small quantity of urine and evaporate to dryness. Dissolve the residue in a test-tube with the aid of a solution of sodium carbonate. Moisten some filter paper with a 10 per cent. solution of silver nitrate. Allow a few drops of the solution in sodium carbonate to fall in the center of the moistened filter paper. If uric acid is present, the silver nitrate will be reduced to black metallic silver.



**Hippuric Acid**, in combination with sodium and potassium, is usually present in the urine of man to the extent of 11 grains (0.7 Gm.). This amount is increased when asparagus, plums, cranberries, apples, grapes, etc., are eaten, or benzoic or cinnamic acids are administered. There is evidence that hippuric acid is formed in the kidney from benzoic acid. The former crystallizes as rhombic prisms, resembling somewhat the "coffin-lid" crystals of triple phosphates. Hippuric acid crystals are distinguished by the fact that they are precipitated in acid urine only, and also by their not dissolving on the addition of acetic acid. The phosphates, on the contrary, are precipitated only in neutral or alkaline urine, and are readily soluble in dilute acetic acid.

**Creatinin**, a crystalline nitrogenous compound, is excreted daily to the extent of 15.4 grains (0.9 Gm.). It is the end product of metabolism of muscle albumen, and its source is the creatin of the muscles of the body and of the muscles of animals eaten as food. It is distinguished in the urine by its union with zinc chloride, with which it forms an insoluble compound, appearing under the microscope as minute needles, arranged as balls and rosettes. Creatinin reduces alkaline copper solutions, and therefore affects, in a slight degree, the accuracy of the quantitative sugar estimations which depend upon the reducing power of sugar-containing urine.

*Weyl's Test.* Add a few drops of a freshly made, very dilute aqueous solution of sodium nitroprusside and a few drops of dilute sodium hydrate solution to about 10 mls (2.7 f3) of urine in a test-tube. In the presence of creatinin a ruby red color appears which changes after a short time to an intense yellow. If this solution be heated with a little glacial acetic acid, the yellow will change to green, and finally to blue. Acetone gives a similar reaction, but on the addition of acetic acid changes to a purplish red instead of green. If the urine be heated previous to the application of this test, the acetone will be driven off.

*Jaffe's Test.* Add a few drops of a saturated watery solution of picric acid and a few drops of 10 per cent. sodium hydrate solution to some urine in a test-tube. If creatinin is present a red color appears immediately, which increases in



intensity and remains permanent for a long time. If glacial acetic acid is added, the color becomes yellow. Acetone gives a reddish yellow color of less intensity than that produced by creatinin. Glucose, if present, gives a red color, especially if the mixture be warmed.

**Inorganic Salts.** Sodium and potassium phosphates, known as the alkaline phosphates, are found in the urine, the total quantity being about 61.7 grains (4 Gms.). Calcium and magnesium phosphates, known as the earthy phosphates, are present to the extent of 15.4 grains (1 Gm.). They are held in solution in the urine by its acid constituents. If the urine be rendered alkaline, they are precipitated. Sodium and potassium sulphates are also present to the extent of about 30.9 grains (2 Gms.). The phosphoric and sulphuric acids, which are combined with these bases, enter the body, for the most part, in the foods, though there is evidence that they also arise by oxidation in consequence of the metabolism of proteins which contain phosphorus and sulphur.

Sodium chlorid, the most abundant of the inorganic salts, is derived mainly from the food, and is excreted to the extent of about 231.5 grains (15 Gms.) in twenty-four hours.

As previously stated, when the urine becomes alkaline, the phosphates are precipitated and appear as coffin-lid crystals of ammonio-magnesium phosphate, needle-like crystals of calcium phosphate, or as amorphous phosphates, *i.e.*, fine granules. The phosphoric sediments are readily distinguished by the alkaline reaction of the urine, and by their insolubility by heat (by which the urates are dissolved) and their solubility in acetic acid. This sediment acquires importance only when formed within the bladder as a result of infection.

**Anomalies and Malformations.** The absence or maldevelopment of one kidney is not uncommon, and its discovery is usually accidental. As a rule, malformation of one or both kidneys is of no great importance. The absence of one kidney, however, may become of the utmost importance when a renal affection requiring operation occurs. The sudden abolition of function of the single organ, as, for instance, from occlusion of the renal pelvis or ureter by a calculus, may cause death. The occurrence of renal colic, with complete anuria lasting for some time, may suggest the correct diagnosis,

which is confirmed by the finding of but one ureteral orifice on cystoscopic examination of the bladder. Very rarely two ureteral orifices are found in the bladder when there is but one kidney.

More frequently the kidneys are fused at either the upper or lower poles forming the horseshoe kidney (*Ren Arcuatus*). Usually the lower poles are fused, while the upper poles are free and lie to the right and left of the vertebral column. Very rarely the kidneys may be fused throughout their length, and both together may lie entirely to the right or left of the spinal column. Such anomaly may lead to errors in diagnosis, as the fused kidneys may be mistaken for a tumor. Their removal would, of course, lead to the death of the patient. The existence of a developmental error, such as absence or maldevelopment of one or both kidneys may be suspected when there is some anomaly of the sexual organs, as the two are often associated, and may be accurately diagnosed by radiograms.

### DISPLACEMENTS OF THE KIDNEY.

Either one or both kidneys may be displaced. The ptosis may be congenital or acquired. The displaced organ, or organs, may be freely movable or fixed in an abnormal location. When the displacement is congenital the left kidney is more likely to be affected, and is said to be more commonly found in men than in women.

Acquired displacement of the kidneys is more common in the female, is usually unilateral, the right being more frequently involved. As a rule, the organ is freely movable. This mobility may vary from a slight degree that permits easy palpation of the lower pole during inspiration, to a degree that permits the organ to be moved by the palpating hand in various directions in the abdomen. It is possible to classify movable kidney into three groups according to the degree of mobility. In Group A, The Palpable Kidney, one is just able to feel the lower pole below the edge of the ribs during deep inspiration only. When the entire organ descends so low that one may dip down between the edge of the ribs and the upper pole of the kidney, it may be called a Movable

Kidney, and is placed in the second group. In the third group, Floating Kidney, the organ is so freely movable that it may be felt just above Poupart's ligament, or in the mid-line of the body. It may even be pushed to the opposite side of the spinal column by the palpating hands.

Mobile kidney is very common, and, as usually encountered, belongs to Group A or Group B. Examples of the third group are much more rarely observed.

The most important *etiologic factor* is sex. The relation of incidence in women to men is given by Dietle as 100 to 1. This corresponds to our own experience, in which men have been found to be very rarely affected. While it may occur at any age, it is most commonly found in adults between the ages of 30 and 60 years. Relaxation of the abdominal wall, such as may follow child bearing, and overdistention from fluid or tumors, causing changes in the intra-abdominal pressure, is a factor. The body form is an important etiologic factor in movable kidney. The space normally occupied by the kidneys, kidney niches or depressions, may be flatter than normal, and on the right side, especially in women, more open below, so that dislocation more readily takes place. Such a condition of affairs is observed in women of slender build with narrow thorax and flattened abdomen.

Tight lacing may cause a dislocation of the kidney by compression of the superior half of the abdomen, forcing downward the viscera; the disturbance of circulation resulting therefrom favors relaxation of the attachments of the kidneys, still further favoring their displacement.

Extreme emaciation with loss of fat from the renal capsule may sometimes permit of very greatly increased renal mobility. A predisposition to movable kidney exists in certain individuals, and other factors may be at work, as renal displacement is frequently not observed in emaciation. The right kidney is most usually affected. Rarely are both kidneys dislocated, and still more rarely the left alone. The fact that the right kidney is more often affected than the left is probably due to the difference between the peritoneal and connective tissue attachments on the two sides of the body. The anterior portion of the renal fascia is reinforced on the left side by a triple layer of peritoneum. The colon probably

lends some support to the left kidney, and ptosis of the splenic flexure is extremely rare. The hepatic flexure of the colon, on the other hand, is very frequently ptosed, and normally there is a greater downward pull exerted by the cecum, because of the weight of fecal material, which is so commonly retained there. The left renal artery is shorter than the right, and is rather closely attached to the pancreas by cellular tissue.

Distortion of the vertebral column, tumors in the region of the kidney, or the weight of a large pleural effusion may displace the kidney downward. Traumatism may occasionally play a rôle. Very commonly, movable kidney is associated with gastroenteroptosis. It may be present as a part of the general ptosis of the abdominal viscera in Glenard's disease.

In the vast majority of instances symptoms are absent, the condition being discovered in the course of a routine physical examination. When thus found, judgment should be exercised in determining its relative importance in the clinical picture presented by the patient, who is often neurasthenic, and sometimes hysteric. Unless it is evident that the mobile kidney is producing symptoms, it is best to keep the patient ignorant of its existence, more especially in the case of a hysterical or neurasthenic subject. Not infrequently individuals are encountered who have become hypochondriacal after being told that the kidney is movable.

As has been stated, movable kidney usually is associated with ptosis of the colon or stomach. Symptoms, such as constipation, recurring headaches, with or without nausea and vomiting, due to autointoxication from fecal stasis; a sense of weight in the region of the stomach after eating or drinking, are due to the ptosed stomach and colon, and have no relationship to the mobility of the kidney. Symptoms directly referable to the movable kidney are sometimes noted, and consist most commonly in a sense of discomfort in the right or left loin, as the case may be, and a dull aching in these regions and in the flanks after standing, lifting, straining or walking.

Patients, in rubbing the abdomen to relieve the aching, sometimes feel the mass which they suppose is a tumor, and

are thereby led to consult a physician. Usually the aching and discomfort disappear when the recumbent posture is assumed, and it is not uncommon to note that these symptoms are complained of especially toward the end of the day. Cases have been reported in which jaundice occurred, due to pressure by the kidney on the gall-ducts. If this does occur, it is extremely rare. It is more probable that the jaundice is coincidental, *i.e.*, due to a cause entirely dissociated from the mobile kidney.

When the mobility of the kidney is considerable a Dietl's crisis may occur, due to a twisting or kinking of the ureter or renal vessels. Sudden, sharp pain occurs in the renal region of the affected side, with chill, fever, nausea and vomiting. The urine becomes diminished in quantity, is high colored, and may contain albumen and blood. Urates and calcium oxalate crystals are frequently found in the urine. Prostration is sometimes very pronounced, and the pain may be as severe as that of renal or ureteral calculus. The abdominal muscles become tense and abdominal palpation painful. The rigid abdominal muscles may preclude the possibility of feeling the kidney. It is sometimes impossible to differentiate between a Dietl's crisis and renal or ureteral calculus. In some cases a mass may be felt which is very tender on pressure, and which gradually increases in size during the attack, due to increasing hydronephrosis. As the attack subsides the flow of urine increases and may suddenly become quite profuse. The pain may disappear rapidly or gradually, and may be followed by a sense of soreness in the renal region. The rigid abdominal muscles gradually relax, and palpation then reveals the displaced kidney, which usually is tender on pressure for several subsequent days. The attack may last from a few hours to a day, or more, and may recur at irregular intervals.

As a rule, the diagnosis is easily made by palpating a movable mass which has the shape and consistence of the kidney. It is felt to descend deeply from beneath the ribs, and usually moves up and down with respiration. It may be painless on pressure, or when compressed between the palpating hands may give rise to a dull, sickening ache. In palpating for a movable kidney the patient should lie, prefer-



ably, on the back with the knees flexed so as to secure the maximum relaxation of the abdominal muscles. In palpating for the right kidney the left hand should be placed against the patient's back just below the last rib on the right side, while the right hand is placed opposite on the abdomen. A deep inspiration will drive the kidney downward between the palpating hands. If this fails, the patient should sit up for a few minutes breathing deeply or arise and walk about the room, after which an examination will be quite successful. It is frequently recommended that the patient be examined while in the erect, or in a stooping posture, but, as a rule, the abdominal muscles are thus rendered too rigid to permit of a satisfactory examination. As stated, the diagnosis during a Dietl's crisis may be very difficult, or even impossible, if the kidney cannot be palpated and the patient is seen for the first time. The condition may be confounded with a constricted hepatic lobe (Schnürlappen), particularly the tongue-shaped prolonged anterior lobe, or with a thickened and enlarged gall-bladder frequently found after repeated attacks of gall-stone colic. There is a notch between such a lobe and the head of the gall-bladder which closely simulates the hilus of the kidney; the lobe can often be turned over so that it apparently occupies the position of the kidney; and the symptoms produced by gall-stones are quite similar to the symptoms of movable kidney. A mistake in diagnosis is, therefore, easy to make. By carefully examining the suspected region by palpation and percussion, with the patient lying on the left side, the relation of the mass to the liver can usually be determined.

#### TREATMENT.

Many cases require no direct treatment. This is particularly true of that large class of cases without symptoms, the mobility of the kidney having been discovered during the course of a routine physical examination. In many instances, the patient, usually a woman, seeks relief from symptoms denoting neurasthenia. Such a patient is likely to be under weight, with a long, narrow thorax, with an acute subcostal angle, a costal margin that dips down laterally, almost touching the iliac crests, and general muscular relaxation, espe-

cially well marked in the abdominal muscles. Formerly these symptoms, *i.e.*, of neurasthenia, were erroneously attributed to the renal mobility. Today it is generally recognized that the latter may be only a detail of the general symptom complex presented by the patient, and is only occasionally the real factor of neurasthenia. The treatment is, therefore, directed to the whole body, and is based upon a study of the particular individual, a study that includes an estimate of the digestion and assimilation of food, intestinal toxemia, and similar details.

To as great an extent as possible the patient must be relieved of responsibility, so as to avoid further fatigue of the nervous system. If the neurasthenia be of sufficient degree, a complete rest cure is necessary. Ordinarily the patient is directed to retire at 8 P.M. or 9 P.M. and to remain in bed until 8 A.M. or 9 A.M. Upon arising a salt water sponge bath should be taken. For this purpose an earthenware vessel containing a saturated solution of rock salt is kept in the bathroom. With this solution the body should be sponged quickly, and this followed by brisk rubbing with a coarse Turkish towel. For one unaccustomed to cold water the sponge baths may be taken at a temperature of 98° F. (36.6° C.) gradually, day by day, reducing the temperature of the water until it is the same as that of the room. If burning or itching of the skin follows the use of the brine, it may be diluted, or the body may be sponged with clear water after the brine sponge bath.

Then follows breakfast, which should consist of fresh fruit, a cooked cereal, one or two poached or soft-boiled eggs, and bread or rolls with butter. Milk or cocoa is preferable to tea or coffee because of their high nutritive value. Dinner should be eaten in the middle of the day, and should consist of soup, meat and vegetables. The evening meal should be light. Meat should be eaten but once daily.

If the examination of the stomach contents obtained one hour after the ingestion of a test breakfast reveals a hypochlorhydria, hydrochloric acid, U. S. P., in 10- to 20- drop (0.62 to 1.2 mls) doses in water, should be given after meals. Pepsin should be supplied when a test shows it to be absent, an

event which is extremely rare. The feces must be examined at intervals to detect the presence of carbohydrate fermentation or protein decomposition, and if either be found the dietary must be modified accordingly. In some instances the administration of pancreatin and diastase has seemed to aid the digestion. The importance of thorough mastication in the preparation of the food for the stomach must be remembered, and if molars are absent, an efficient artificial grinding surface should be supplied by a dentist.

As a part of the general muscular relaxation, atony of the muscular coat of the colon, especially of the cecum and ascending colon is present. This permits of fecal stasis, during which as a result of bacterial activity, decomposition of proteins or carbohydrates with the formation and absorption of toxic substances occurs. A study by means of the X-rays will, of course, establish beyond question the diagnosis of fecal stasis. Clinically, this may be determined as follows: The bowels are permitted to move spontaneously at the usual time in the morning after breakfast; or, if necessary, an enema of 1 pint (473.1 mls) of normal salt solution is given, so as to empty the rectum and sigmoid flexure. Immediately after the bowels move a soft rubber rectal tube is inserted 6 to 8 inches (15.2 to 20.3 cm.) into the rectum, and 3 pints (1.5 l.) of warm normal salt solution (a teaspoonful (3.7 mls) of table salt to 1 pint (473.1 mls) of water) is allowed to flow slowly into the rectum while the patient lies on the right side. Sometimes, owing to an accumulation of gas in the sigmoid or rectum, or to a failure of the solution to pass into the descending colon, the patient experiences a strong impulse to evacuate the water, in which event the inflow should be stopped and the abdomen rubbed, with deep pressure, from below upward to the costal border on the left side. In a few seconds the spasm disappears, and more of the solution is allowed to flow in. It may be necessary to resort to the manual dispersion of the liquid in the colon several times before 3 pints (1.5 l.) are finally introduced. When all the salt solution has entered the colon, the rectal tube is withdrawn; the patient then lies on the back and the abdomen is rubbed from below upward to the costal border on the left side, from left to right across the abdomen along the usual

position of the transverse colon, and from the costal margin downward on the right side. In this manner the solution is driven into the cecum, and its presence there is determined by a splashing sound on succussion over the right lower abdominal quadrant. The water should then be evacuated into a jar, so that the amount of fecal material returned with the salt solution may be estimated. More than 4 ounces (118.4 mils) of feces, by volume, should be regarded as evidence of fecal retention, and should be treated by colonic lavage, the technic of which is identical with that of the diagnostic lavage just described.

The presence of indican in the urine, due to the decomposition of proteins, is very commonly associated with fecal stasis, and often indicates the success or failure of efforts to empty the colon or cecum. Indican may be tested for very easily and quickly as follows: To 3 or 4 mils (48.6 or 64.8 *m.*) of urine add 1 drop of 1 per cent. solution of potassium chlorate, 2 or 3 mils (32.5 or 48.6 *m.*) of chloroform, and as much strong hydrochloric acid as urine. Thoroughly mix by pouring from one test-tube to another eight or ten times. The appearance of a blue color in the chloroform, which falls to the bottom of the tube, denotes the presence of indican. A rough idea of the quantity may be formed by the depth of color.

The lack of muscular tone should be treated by general massage, which is later replaced by resisted movements and active exercise, especial attention being directed to the abdominal muscles. The following exercises are recommended:

(a) Lying flat on the back, body in a straight line, toes turned upward and arms extended by the sides, the head is slowly raised, bending the neck until the chin touches the chest. Then the trunk is slowly raised until the body is brought to a sitting posture. While this is being accomplished the heels must remain in contact with the floor, the knees kept straight and stiff, and the hands allowed to slide along the thighs. The body is then slowly lowered to recumbency.

(b) Lying flat on the back with the body in a straight line, toes turned upward and arms extended by the sides, the legs are raised slowly from the hips, keeping the knees stiff,



until at right angles to the trunk. They are then slowly lowered to the floor.

These exercises must be practised cautiously. It may be necessary to advise the patient to raise the body a few inches only from the floor, and not to attempt to assume the sitting posture, because of the possibility of straining the muscles or producing a hernia. Supervision is therefore necessary.

Where the abdominal muscles are extremely atonic, and in cases of mobile kidney secondary to multiple pregnancies, a binder with a pad is necessary. The binder may be of silk or cotton, supplied with perineal straps to prevent it from slipping upward, and should come well down on the hips. A pad may be used, and must be so placed as to make pressure upward and backward and toward the right, in order to push the kidney towards its normal position. The pad should be soft but firm, about 3 inches (7.6 cm.) long and 2 or 2½ inches (5.08 or 6.3 cm.) wide. It is so placed that the upper border faces upward and towards the right, lying a little below the line passing from the umbilicus to the anterior-superior spine of the ilium. The belt should always be applied while the patient is in the recumbent posture, and the kidney is in place. It should never be applied while the patient is in the erect posture, as the pad may then be above the kidney, and by pressing downward, thus aggravate the condition. The wearing of a binder should be looked upon as a temporary measure, to be used until the emaciation is overcome, or until the abdominal muscles become strengthened by exercise. In extreme cases it is necessary to take up the redundancy in the overstretched abdominal muscles by surgical means.

The anchoring of the kidney surgically should never be done excepting in those cases of floating kidney that give rise to Dietl's crisis, or show evidence of congestion due to twisting of the renal vessels; also when other measures have failed to relieve cases in which neurasthenia can be definitely ascribed to the movable kidney.

## ANOMALIES OF URINARY SECRETION.

**Anuria.** Total suppression of urine occurs in intense congestion of the kidneys, which may be active, as in acute



nephritis due to infections, fevers, poisoning with phosphorus, arsenic, mercury and lead; or passive, as in the cyanotic kidney, which occurs in cardiac dilatation and late in the course of myocardial degeneration. The anuria may be mechanical, as the result of blocking of the ureter by a calculus, blood-clot or a thick mass of pus, or by the compression of the ureters by new growths in the abdominal cavity. It may follow ureteral catheterization, and is sometimes seen in hysteria.

A patient may live for days, and even for one or two weeks, with complete suppression of urine, during which time consciousness may be retained and the mind remain clear. Nausea and occasional vomiting may occur. Convulsions may not occur. In a fatal case of bichlorid of mercury poisoning, observed by the authors, complete suppression of urine existed for a week, during which time there were no symptoms of uremia. It is necessary to differentiate between anuria or urinary suppression and retention. In any disease characterized by extreme adynamia, as in the typhoid state, or when unconsciousness occurs, or when opium is given, the kidneys may secrete a greatly diminished quantity of urine which is retained in the bladder. In hospital practice it is not uncommon to find, on admission, that the bladder of the patient who had passed no urine for a day or two contains 16 to 40 (473 to 1183 mls), or even 50 ounces (1478 mls) of urine. It is, therefore, necessary, in the case of all patients who are thought to be anuric, to palpate in the suprapubic region for a distended bladder; and, if necessary, to resort to catheterization to establish diagnosis. Treatment depends upon the nature of the underlying cause. Thus, when the anuria is due to obstruction of the ureter or ureters, surgical intervention is necessary. Occurring in the course of nephritis, efforts must be made to increase elimination through the skin and bowels. To increase elimination through the skin, sweating induced by heat is the safest method, and may be accomplished by the use of hot wet packs, hot dry packs, or electric light baths (see page 47 for technic). The use of pilocarpin is dangerous in many cases, and when employed the patient must be closely observed, because of the possibility of pulmonary edema.

Elimination through the bowels should be secured by sodium sulphate, or sodium phosphate, or both. An effort should be made to secure six or eight watery bowel movements in twenty-four hours.

If there is no edema (and there is likely to be none if the anuria is due to metallic poisons, such as phosphorus or mercury) 2 ounces (60 mls) of water, either hot or cold, should be given every hour. If edema occurs, the amount of water consumed should be reduced to a half or a third of this amount.

No food except skimmed milk or whey should be given until the kidneys begin to secrete. Because of the speed with which the milk is carried through the gastrointestinal tract, due to the administration of cathartics, the milk should be peptonized, so as to reduce the necessary time for digestion as much as possible.

Diuretics, such as squill, diuretin, Basham's mixture, and the like should not be given. If there is evidence of cardiac distress the infusion of digitalis, made from assayed leaves, may be given in from 2- to 4- dram (7.5 to 15 mls) doses every four hours. The application of heat, poultices and dry cup to the loins sometimes seems to be of benefit. When the anuria occurs during acute infectious diseases, or after operation, hot rectal enemata of coffee are of value.

**Hematuria.** As the term indicates, blood is present in the urine. It is a symptom and not a disease entity. The quantity of blood may be so small as only to impart a faint tinge to the urine, or it may be so large as very greatly to change the color. Depending upon the quantity of blood and the age of the urine, the color may vary from a light smoky tint to a light red or a dark color resembling porter. The color may change from brown to pink on standing exposed to the air. The brown tint is usually due to the presence of methemoglobin.

The hemorrhage may come from any portion of the genito-urinary tract. Renal hemorrhage may be due to tuberculosis, carcinoma, hypernephroma, polycystic degeneration, acute nephritis, especially that caused by turpentine, hexamethylene-tetramin (urotropin), carbolic acid and cantharides; infarcts, calculus pyelitis, traumatism, as from contusions,

stab wounds, gunshot wounds, rough palpation of a movable kidney and parasites, such as the distoma hematobium and the *filaria sanguinis hominis*.

Ureteral hemorrhage may result from carcinoma, from traumatism such as may occur during operations, or from the passage of a calculus.

Vesical hemorrhage may be due to benign or malignant papilloma, carcinoma invading the bladder from surrounding structures, calculi, acute cystitis, and varicose veins at the vesical neck.

The source of the bleeding may be urethral, due to rough instrumentation, passage of calculi, and traumatism from external violence. Bleeding may occur during the course of a severe gonorrhea.

Hematuria is sometimes observed in purpura, scurvy, malaria, leukemia and hemophilia. The presence of large quantities of uric acid crystals, and more especially, crystals of calcium oxalate may cause slight hematuria. Renal hematuria, only discoverable microscopically, is usually found in septic nephritis and congestion. In some cases no cause can be found for the bleeding, even on operation. Such a condition is then designated as essential hematuria, idiopathic hematuria, renal epistaxis and renal hemophilia. The bleeding comes from one or both kidneys, without discoverable adequate exciting cause, and may be associated with pain. It occurs usually in patients under 30 years of age.

The recognition of hematuria is very easy. The color of the urine, however, is by itself not diagnostic, as it may be produced by other pigments. Furthermore, by the color alone it is impossible to distinguish hematuria from hemoglobinuria. The finding of numerous erythrocytes in the urine by microscopic examination settles the diagnosis. The erythrocytes may retain their color and shape, or they may be crenated or appear as rings without color (shadow corpuscles). Spores and oil globules may be confusing, but a very little experience enables one to recognize them by their refractiveness and great variability of size.

The discovery of the source of bleeding may be very difficult, and at times impossible. In cases of renal hemorrhage the urine usually contains casts of various kinds, including

blood-casts. The blood and urine are thoroughly mixed, and do not readily separate on standing. If the urine is voided in two portions, both will be equally bloody. If the blood comes from the urinary passages, it is more readily separated by sedimentation. When the source of bleeding is the bladder, the first portion of urine voided may be clear and the last bloody. The blood may appear at the end of micturition. In washing the bladder the water returns blood-tinged, while if the blood comes from the kidneys or ureters, the water will return clear. When the urethra is the source of bleeding the first portion of urine voided will contain the blood; the second portion may be clear. These points are not very reliable, however, and a cystoscopic examination should be made. This procedure is always necessary in renal hemorrhage to determine whether the blood comes from the bladder or from one or both kidneys. Differential ureteral catheterization determines from which kidney the blood comes.

As hematuria is but a symptom, it is necessary to seek and treat the underlying disease. When the hemorrhage is severe, rest in bed must be insisted upon, and continued until the evidence of blood disappears from the urine. The diet should be bland, consisting of foods rich in purin bases, and such vegetables as rhubarb, which cause oxaluria, should be avoided. As highly concentrated urine is very irritating, the specific gravity must be kept low by the drinking freely of water. Pituitrin, adrenalin, calcium lactate may be given internally as hemostatics. Hexamethylene-tetramine (urotropin) in large doses is recommended by some authors, but it must not be forgotten that this drug, even in moderate doses, may produce hematuria. In essential idiopathic hematuria, when the hemorrhage is severe and long-continued, good results have been reported from exposing the kidney and incising the organ. Why the hematuria disappears in these cases is unknown. The appearance of blood in the urine may greatly excite the patient, in which event it may be necessary to administer bromides, or even morphine.

**Hemoglobinuria.** By this is meant the presence of hemoglobin or methemoglobin in the urine. Up to a certain point, hemoglobin released from the erythrocytes, destroyed in the body is converted into bile pigment. When, however, hemo-



lysis of a large number of erythrocytes suddenly occurs the hemoglobin is excreted by the glomeruli in the kidneys.

Hemoglobinuria occurs after poisoning by potassium chlorate, carbolic acid, pyrogallie acid, naphthol, phosphorus, arsenuretted hydrogen, carbonic dioxid, toulene-diamene, and after the ingestion of poisonous fungi or of tainted edible mushrooms. Sometimes it also occurs in scarlet fever, typhus and typhoid fevers, yellow fever, syphilis, scurvy, purpura and malaria. In the latter disease it is commonly seen in hot climates, where it is termed malignant malarial hemoglobinuria, and in Africa is called black water fever. It may also occur after extensive burns, during absorption of extensive hemorrhagic effusions, after violent exercises, and after the transfusion of blood of another animal of different species. The hemoglobinuria of newborn children is probably the result of infection.

There is a peculiar type—paroxysmal hemoglobinuria—the etiology and pathology of which are unknown. It occurs in persons apparently otherwise healthy, and in the majority of instances the exciting cause appears to be exposure to cold. That some other factor is also present is evidenced by the fact that the disease is rare, while the number of individuals exposed to very low temperature is great. The attack comes on suddenly, and may be preceded by chills and fever. Vomiting and diarrhea may be very prominent symptoms, and aching, and even pain, in the lumbar regions is rather common. Cyanosis of the ears and tips of the fingers may appear during the attack. A paroxysm may occur several times in one day and then cease; or one may occur on several successive days.

When the hemoglobinuria is slight the urine may be pink and transparent. In severer cases the urine has a deep red or brown color, approaching to blackness. On standing, a very dark, sometimes chocolate-colored sediment, is deposited, which is found to be composed of amorphous brownish granules, sometimes arranged in cast-like formation. A characteristic that distinguishes hemoglobinuria from hematuria is the absence of erythrocytes; or, if present, they are so few as to be negligible. Urates and calcium oxalate crystals may be present in abundance. The urine during the attack always



contains albumin, which may persist for a time after the hemoglobin or methemoglobin disappears.

While the underlying cause of hemoglobinuria may be difficult, or even impossible to determine, the recognition of the presence of the hemoglobin in the urine is easy. It must be remembered, though, that a similar appearance of the urine may be imparted by urobilin and hematoporphyrin, which is a pigment very similar in composition to hematin. The presence of hemoglobin can be detected by Heller's blood test, which is carried out as follows:

To a test-tube half full of urine 5 drops (0.31 mils) of potassium or sodium hydroxid are added and the mixture heated. If hemoglobin is present, a brownish red or blood red flakey precipitate appears. It consists of the phosphates and carbonates of the earthy alkalies which have carried down with them the hematin that has been formed from the hemoglobin in the reaction. In alkaline urines the above method often produces no precipitate, because the phosphates and carbonates have already completely separated out spontaneously. The necessary quantity of phosphates and carbonates may be supplied by adding to the specimen about the same volume of a normal urine. With this test the coloring matters which appear in the urine after the use of chrysarobin, senna, rhubarb or cascara sagrada, may react very much like hemoglobin, and so may lead to confusion. But in the latter case the red color addition of an alkali after cooling, and the decoloration upon the addition of acetic acid, are characteristic.<sup>1</sup> Another very delicate test is the Turpentine-Guaiac Test, which is performed as follows: A layer consisting of a mixture of equal parts of tincture of guaiac (alcoholic solution of resina guaiac 1:5) and oil of turpentine is carefully stratified upon the top of the urine. If hemoglobin is present a cloudy ring slowly forms at the junction of the two layers, gradually becoming an intense blue. The oil of turpentine must be ozonized, *i.e.*, old. The urine must be acid. If alkaline, acidulate with acetic acid.<sup>2</sup>

Examined spectroscopically, there are either the two absorption bands of oxyhemoglobin, which is rare; or, more commonly, there are the three absorption bands of methemoglobin, of which the one in the red near C is characteristic.

If the hemoglobinuria has been caused by drugs, their use must be discontinued. Its incidence during the course of malaria prompts one to determine whether or not the symptom is due to the great destruction of the erythrocytes by the plasmodium, or is referable to excessively large doses of quinin. When attributable to syphilis, active antisiphilitic treatment is indicated. It is probable that a hemolytic toxin is the cause of some hemoglobinurias, and in the absence of more definite etiology attempts should be made to secure free elimination through the skin, bowels and kidneys. For this purpose hot wet packs or electric light baths (see page 584 *et seq.*) should be employed, three or four watery evacuations of the bowels secured, and either normal salt or a 2 per cent. dextrose solution administered by the method of continuous enteroclysis (see page 583 *et seq.*). Arsenic is sometimes of value, and the inhalation of amyl nitrite is recommended by Chvostek, who claims to have been able to abort attacks of paroxysmal hemoglobinuria in some of his patients. In the paroxysmal type of the infection the patient should be kept warm, and water should be given freely.

**Chyluria**, or the occurrence of chyle, in the urine is a rare condition, and may be parasitic or non-parasitic in origin. In the tropics the parasitic variety is more common, and is caused by the filaria Bancrofti, which lodge in the lymphatics. There is usually a dilatation of the lymph-vessels in the kidneys and in the abdominal plexes. The non-parasitic form is extremely rare, and the real causation is unknown. The urine is of an opaque white color resembling milk. If blood is admixed, the color will be purplish or pale red. The freshly voided urine is faintly acid or neutral. Albumin is always found. The microscope reveals many minute fat droplets. In some instances coagula form in the urine, either after voiding or in the bladder. In the latter event, micturition may be very painful.

The course and duration of chyluria are extremely variable. The disease may last for months, or even years, during which time long intervals occur in which the urine appears normal. Almost all cases spontaneously recover.

No effective treatment is known for the non-parasitic variety. When filaria is the cause, the treatment must be

directed to the destruction or removal of the parasites from the body, and prophylactic measures must be employed to prevent reinfection, and the infection of healthy persons.

**Albuminuria**, or the presence of albumin in the urine in quantity sufficient to be detected by the ordinary tests, is always abnormal. The significance of its presence depends upon the cause and the source from which it came. When it is associated with organic changes in the kidneys it is of the greatest importance. The finding of albumin in the urine makes it necessary that the cause be thoroughly investigated. Not all albuminurias, however, are of importance; and it must be emphasized that the discovery of albumin does not make a diagnosis of nephritis. Not infrequently patients are seen who have been made neurasthenic, or even hypochondriacal, by the erroneous importance given to the accidental discovery of albumin in the urine. Albumin may be added to the urine as a result of disease in the genito-urinary tract below the level of the kidneys, such as inflammation, especially with pus formation, in the ureters, bladder or urethra. It may be present as the result of contamination of the urine with pus or blood from the vagina.

Albuminuria is present in very many cases of fever, and is spoken of as febrile albuminuria. It has no serious significance, and disappears when the temperature returns to normal. It is probably the result of a cloudy swelling of the renal epithelium due to the toxin that caused the fever. A few hyaline casts are often in association with this form of albuminuria.

In diseases associated with profound changes in the blood, such as scurvy, purpura, leukemia, pernicious anemia, albumin may be found in small quantities in the urine. It is not uncommonly found in severe anemia secondary to carcinoma or sarcoma. Chronic poisoning with lead, mercury, alcohol, etc., will cause albuminuria. It is probable that the albumin from this cause, however, is the expression of permanent organic change in the kidneys. When jaundice occurs albuminuria is very often associated, due either to the irritant action of the bile on the kidney cells or to associated toxins. Albuminuria may be the result of renal congestion, as in cardiac disease.

Functional albuminuria, by which is meant the appearance of albumin in the urine of adolescent individuals whose kidneys are normal as far as can be determined and whose general health is good, is sometimes seen. It is usually intermittent, and is apt to appear after the patient has been in the upright posture for some time. It may also occur after exercise or cold bathing. In one case observed the albuminuria was constant, and was increased by cold baths and exercise. No evidence of renal disease could be found, and the general health of the young man was good. Ether anesthesia for an appendectomy brought out no evidence of renal insufficiency. The absence of albuminuria does not eliminate a possibility of Bright's disease.

Bence-Jones proteinuria, by some referred to as albumosuria, is an albuminous substance which is foreign to the blood, and hence readily eliminated by the kidneys. It is found in cases of multiple myeloma. Its origin is unknown. It is not an indication of disease of the kidneys. The quantity which appears in the urine is variable, but when present is usually continuous. Food seems to have no influence on the quantity of the protein excreted. Its detection in the urine is easy if we remember that, unlike ordinary albumin, it coagulates at a very low temperature. It is, therefore, thrown out of solution, causing turbidity when the urine is heated to 122° to 140° F. (50° to 60° C.). Raising the temperature to a higher degree causes a disappearance of some of the turbidity. It may be tested for as follows:

Acidulate the urine with acetic acid and add a small quantity of concentrated sodium chloride solution. Heat the urine to 122° F. (50° C.). If the Bence-Jones protein is present, the urine becomes milky. Heat still further; at 140° F. (60° C.) a tenacious precipitate is thrown out, tending to adhere to the sides of the tube, and forming a granular mass which floats on top of the urine. As the boiling point of the urine is reached, the precipitate and the cloudiness disappear, reappearing when the urine cools.

**Tests for Albumin.** In a rough way albumin may be detected as follows: Pour urine into a test-tube until it is about three-quarters full. Heat the upper part of the urine. If turbidity occurs it may be due to phosphates, carbonates

or albumin. If the cloudiness disappears upon the addition of a weak acetic acid (1 to 9), it is due to phosphates or carbonates; if the cloudiness increases on the addition of acid, it is due to albumin.

*Heller's Test.* Put about  $\frac{1}{2}$  inch (1.2 cm.) of urine in the test-tube. With a pipette allow nitric acid to run slowly down the inside of the tube. The acid, being much heavier than the urine, will go to the bottom. If albumin is present, a white ring appears at the margin of contact of the two liquids. The density and depth of the ring depends upon the amount of albumin present. The nitric acid may be placed in the tube first, and the urine carefully poured on top by allowing it to run down the side of the tube. The ring of turbidity may be due to the presence of urates or uric acid, in which event it will disappear if the contents of the tube is warmed (not boiled). After the taking of certain of the balsams the addition of nitric acid to the urine may cause a precipitate of resinous acids. It is therefore advisable to control the Heller's test by boiling a few mils of urine in another test-tube and adding one-third volume of nitric acid; any cloudiness must then be due to albumin, since the resinous acids could not be precipitated because of the heat and the considerable excess of nitric acid.

When a large amount of albumin is present, the ring of contact is very readily seen. When, however, but a very faint trace of albumin is present, unless the tube is viewed against a dark background, the very faint ring may be entirely overlooked. In the writer's laboratory the testing for albumin is done before a window, the glass of which is painted black except for a strip about  $\frac{1}{2}$  inch (1.2 cm.) wide on the level of the eye of the examiner. Through this narrow strip of white glass enough light is admitted to illuminate the test-tube properly, while the remaining portion of the black glass makes a perfect background, against which even the faintest whitish ring of albumin is easily seen.

*Quantitative Determination.* At the present time there is no accurate method that is applicable by the average clinician. The one most in vogue is the Esbach test. The apparatus consists of a glass test-tube with a rubber stopper. Near the top of the tube is a mark designated R, lower down one desig-



nated U, and below this a series of graduations from 1 to 12, from below upward, constituting the scale from which is read the percentage of albumin by volume per 1000 parts of urine. The reagent used consists of picric acid, 10 Gms. (154.3 grs.); citric acid, 20 Gms. (308.6 grs.); water, 1000 mls (33.8 f $\bar{3}$ ). In making the test fill the tube (Esbach's Albuminometer) with urine to the line marked "U" and raise the level of content to the mark "R" by the addition of the reagent. The tube is then closed with the rubber stopper, and the reagent and urine thoroughly mixed by repeated inversions of the tube without shaking. Then set the tube aside in a strictly vertical position for twenty-four hours, after which the height of the precipitate in the tube is read off on the scale, the results being expressed in terms of parts per 100 or per 1000. When expressed in percentage we must remember and make clear that we mean per cent. by volume and not by weight. The percentage by weight can be determined accurately by precipitating the albumin, drying and weighing the precipitate.

*Casts and Cylindroids.* Casts are found in the tubules of the kidney. Their composition is unknown, but it is supposed that the material of which they are composed is of a protein nature. If the urine of healthy individuals is allowed to stand for some hours in a conical sedimentation glass and the sediment centrifugated, a very few hyaline casts may sometimes be found, even in the urine of young adults. Their discovery in the urine of individuals past middle life has no serious significance when the casts are very few in number, and all other evidence of renal disease absent. Under such circumstances their appearance is probably physiologic. When casts are in sufficient number to be found with ease, they are evidence of more than physiologic change in the kidneys, but do not necessarily mean Bright's disease. They may be found in the urine in renal congestion from any cause, and also in renal irritation, as in oxaluria and in marked lithuria. Bile appears to be a decided renal irritant, and in almost all cases of jaundice, casts, which are usually bile-stained, are found. The administration of mercury in medicinal doses is sometimes followed by the appearance of hyaline casts in considerable numbers, and is due to the fact that mercury is a direct poison to renal cells. In cases of chronic interstitial

and chronic parenchymatous nephritis we have observed the appearance of casts in excessive numbers after the giving of calomel. As stated, the appearance of an occasional hyaline cast in an otherwise normal urine excreted by an apparently normal person has no significance. This is important to bear in mind in connection with life insurance examinations. If, however, a few casts are regularly found in the urine of an apparently normal individual, even though albumin is absent, the kidneys must be suspected of disease, and every means in our power must be used to discover the factor at work; for when we stop to consider how insidious is the onset of chronic interstitial nephritis and that by the time symptoms attract attention to the disease, the prognosis is very grave, the importance of finding the cause of the persistent cylindruria becomes obvious.

Hyaline casts are the most common variety. They are transparent, colorless, and often difficult to see, especially if the microscopic field is brightly illuminated. They vary in length and diameter. The ends may be rounded, flat or irregular, due to fracture. They are found in all types of nephritis, and may be found in any form of renal disease, alone or in association with others. Usually they are present in urine from patients with jaundice.

Granular casts are variable in length and diameter. As a rule they are somewhat wider than the average hyaline cast, especially when observed in parenchymatous nephritis. The granules may be highly refractile, and may be composed of droplets of albumin, fat, and the *débris* of disintegrated epithelium. They are more usually found in chronic Bright's disease.

Epithelial casts are composed of a hyaline matrix, imbedded in which are epithelial cells derived from the tubules of the kidneys. They are usually seen in acute parenchymatous nephritis. They have a greater diameter than the hyaline or granular casts.

Leucocytic casts are composed of a hyaline matrix in which are imbedded leucocytes. They indicate an active inflammation of the renal substance, and are often found in suppuration of the kidneys.

Fatty casts resemble granular casts. They are differentiated by the high refractive power of all, or nearly all, the granules. They indicate advanced degeneration of the renal parenchyma. They are usually found, with epithelial casts, in chronic parenchymatous nephritis.

Amyloid or waxy casts resemble the hyaline casts, but are larger and opaque instead of transparent. They are supposed, by some observers, to represent the end-product of the complete degeneration of renal cells. They are usually found in chronic parenchymatous nephritis.

Cylindroids are allied to the casts, but are less significant, indicating simply irritation of the renal tubules. Their appearance is extremely varied. Some are thin, almost thread-like bodies, faintly striated and of variable length, sometimes stretching entirely across the field of the microscope. Some are ribbon-like, tortuous and bent. Some resemble hyaline casts, except that one end tapers to a point. The chief characteristics of cylindroids are the striations and their tapering ends. Enmeshed in their structure may be uric acid or calcium oxalate crystals and urates. They are seen in large numbers in urine containing an excess of calcium oxalate crystals. They are found in the renal tubules, and their appearance in the urine bears no relationship to albuminuria.

### CIRCULATORY DISTURBANCES OF THE KIDNEYS.

**Anemia.** Anemia of the kidneys may be a part of the general state of the blood or may be purely local, due to narrowing of the lumen of the blood-vessels through the action of the nervous system or to compression of the renal artery by new growths, adhesions or the like. The anemia produced by vasoconstriction is of comparatively short duration, and is observed in hysteria, and as a result of great irritation of one kidney or ureter by a calculus, urethral catheterization, operation on one kidney or traumatism.

When vasomotor in origin, the anemia is transitory and leads to no permanent change in structure. When caused by arterial compression it may be sufficiently prolonged to cause parenchymatous degeneration.

As part of a general process, renal anemia is present in pernicious anemia, and in those diseases characterized by a marked secondary anemia, as advanced carcinoma, tuberculosis, and the like. Under these conditions the anemia is permanent, and leads to degeneration of the renal cells with resulting diminution of function. A result of renal anemia, regardless of cause, is diminution in the quantity of urine excreted, or even complete anuria.

The vasomotor type of anemia is of such short duration that treatment is unnecessary. When anemia is due to compression of the renal artery, surgical interference is requisite to remedy the condition. When due to changes in the blood, the primary disease must be treated. It is obvious that treatment directed to the kidneys must fail if the disease producing the secondary anemia is incurable.

**Hyperemia.** The kidneys may be the seat of active or passive hyperemia. As in other organs of the body, a certain degree of hyperemia is physiologic. Any organ that is actively functioning contains more blood than during quiescence, and the greater the functional activity, the greater will be the blood-supply. The kidneys, being among the principal organs of elimination, are called upon from time to time to remove from the body irritants which have gained entrance through the mouth, or which have been formed in the body, either through faulty metabolism or bacterial activity, as in the infectious fevers. Active hyperemia, therefore, is present in most of the general infections, particularly those characterized by fever, in various disturbances of digestion and metabolism, notably those resulting in great elimination of calcium oxalate and uric acid crystals, and as the result of the taking of such irritant drugs as turpentine, copaiba, cantharides, cubeb, hexamethylenetetramin (urotropin), etc. This state may also be brought about by the use of diuretics. Active hyperemia occurs in one kidney when its fellow is removed by surgery or disease. There is a close connection between the vessels of the skin and those of the kidney, so that chilling of the skin will speedily result in active renal hyperemia.

When the hyperemia is of sufficient grade, the kidneys are swollen. The tension on the capsule may cause a dull

aching sensation in the lumbar regions, and the urine, which at first was increased in amount, diminishes. Albumin, casts, erythrocytes and, in extreme cases, macroscopic blood may appear. The treatment consists in the recognition of the cause. When due to irritant drugs these must be immediately discontinued. Often this is all that is necessary. When the hyperemia is extreme, marked improvement follows the use of hot, dry, or wet packs (see page 584 *et seq.*) to promote sweating, the application of dry or wet cups to the lumbar region and the administration of a saline cathartic. Mercury as a laxative should not be employed. An effort should be made to secure five or six watery evacuations from the bowels. As the blood-vessels of the kidney are already overfilled with blood, but small quantities of water should be given until it is evident that elimination through the kidneys is increasing, when the intake of water should be increased.

The prognosis is good. It must be remembered, however, that if the congestion is long continued, organic changes may occur, permanently damaging the kidneys.

**Passive Hyperemia.** This condition is usually encountered as a part of a general venous congestion secondary to cardiac insufficiency. It may occur late in the course of pulmonary emphysema and fibroid phthisis. Ascites, tumors compressing the renal veins, a pregnant uterus, and thrombosis of the inferior vena cava, are among the causes of passive congestion. Evidence of passive congestion of the kidneys occurring in ascites is one of the indications for the performance of paracentesis abdominis.

The kidneys enlarge and feel tough and firm. They are deep purple, smooth, and the capsule strips easily. The stellate veins are prominent. On section the striations are unusually well marked, the pyramids are deeply reddened, and the blood-vessels and the glomeruli stand out prominently in deep red. Microscopically, the capillaries, especially the glomeruli, are distended with blood, and there may be some coagulated fluid in Bowman's capsule or hyaline casts in the tubules. The tubular epithelium may show some cloudy swelling, especially when the passive congestion is of long standing, in which event the interstitial connective tissue may be increased. When sufficiently long continued, contraction



of the connective tissue may occur, causing a diminution in the size of the organs and leading to changes similar to those of a diffuse nephritis.

Symptoms arising from passive hyperemia of the kidneys are too insignificant to be distinguished among those that are due to the causative factor, such as cardiac insufficiency, pulmonary emphysema, or cirrhosis of the liver. It is detected by the observance of changes in the urine, which consist in the excretion of a greatly diminished quantity of dark color and high specific gravity, which may be over 1030.

The water of the urine is diminished very much more than are the solid constituents; consequently upon standing for a short time after voiding, the urates are precipitated, falling to the bottom of the vessel as a heavy pinkish sediment. If the urine is shaken it becomes opaque. Usually a small amount of albumin is present, with a few hyaline casts and erythrocytes. Granular casts are rarely found in uncomplicated cases. Though the functional activity of the organs is reduced, uremia does not supervene in the absence of a pre-existing nephritis. Occurring during the course of a nephritis, the diminution of function may be sufficient to induce uremia.

When the patient is being studied for the first time it may be impossible to differentiate passive congestion of severe degree from nephritis, especially as in some cases of cardiac decompensation the circulatory disturbance in the brain and gastrointestinal tract may originate the mental changes and vomiting so often seen in uremia. The pronounced cardiac changes with the cyanosis of the fingers, toes, lips, ears, etc., may lead one to the correct interpretation. The diagnosis is confirmed when, under appropriate treatment directed to the heart successfully, the urinary changes disappear. The presence of red blood-cells in the sediment is an important diagnostic sign of renal congestion.

There is no treatment indicated, directed especially to the passive congestion of the kidney, as the condition can be relieved only when the disease that produces the general passive congestion is made to disappear. When the renal congestion is due to pressure on the renal veins by ascites or tumor these must be removed. Occurring late in pregnancy, it need cause no alarm in most cases, and does not, in itself, necessitate abortion.

## NEPHRITIS.

By nephritis is meant a non-suppurative inflammation of both kidneys, first clearly described by Bright about 1827, and since that time usually spoken of as Bright's disease. The term Bright's disease today takes in all the forms of nephritis, although Bright described but one form, namely, an inflammation of the kidneys associated with marked albuminuria and edema or dropsy.

Numerous attempts have been made since 1827 to construct a classification in which all the forms of Bright's disease could be placed, but thus far no satisfactory grouping of the various lesions has been made. Based upon the morbid anatomy the following types are described in textbooks of pathology:

Acute and chronic glomerulonephritis, acute and chronic tubular nephritis, acute, subacute, chronic interstitial nephritis, and arteriosclerotic disease of the kidneys. Clinically, these distinctions cannot be made, and at times it is difficult and even impossible for the pathologist to say from his examination to which type the kidney belongs, for the reason that changes in all the parts of the kidney may be present in equal degree. While in almost every nephritis changes are found in the glomerules and tubules, as well as in the interstitial tissue, the disease is classified according to the location of the preponderating changes. The best clinical classification is that of Senator,<sup>3</sup> as follows:

1. Acute Nephritis.
  - (a) Parenchymatous Nephritis (tubular and glomerular).
  - (b) Diffuse Nephritis.
2. Chronic Diffuse Nephritis without Induration (chronic parenchymatous nephritis).
3. Chronic Indurative Nephritis (contracted kidney).
  - (a) Primary Induration (chronic interstitial nephritis).
  - (b) Secondary Induration (secondary to acute inflammation).
  - (c) Arteriosclerotic Induration.

**Acute Parenchymatous Nephritis.** By this is meant an acute inflammation or degeneration of both kidneys, in which the glomerules, tubules, and often the interstitial tissue, as well, are implicated. The lesions are most usually the result of an infection, and may develop during the course of scarlet

fever, diphtheria, influenza, typhus fever, typhoid fever, acute infectious arthritis (rheumatism), and septicemia from any focus. The renal degeneration or inflammation may be the result of bacterial toxins circulating in the blood, or of the direct action of bacteria upon the renal structure.

Certain drugs and chemicals are capable of causing an acute nephritis. Among them are mercury, lead, cantharides, turpentine, carbolic acid, salicylic acid, potassium bicarbonate, chloroform, ether, and potassium chlorate. Acute nephritis may occur in ptomaine poisoning; and it is probable that highly toxic substances generated in the body as the result of perverted metabolism or the failure of certain organs, as the liver, to destroy poisonous bodies formed during the process of metabolism, may be the factor of some cases of Bright's disease.

As a rule the kidneys are larger, heavier and more vascular than normal. The capsule is thin, glistening, tense, strips easily, and in some instances, when incised, the kidney substance bulges through, giving evidence of the great pressure beneath. The color of the kidneys is dark red in the early stages; but if the disease has been of sufficient duration to permit fatty degeneration to occur, the color is much lighter, and may be a grayish red. If the lesions are chiefly in the glomerules, these appear on section of the organ as dark red points against the grayish-red background.

Microscopically, the predominant lesions may be found in the glomerules (glomerulonephritis), in the tubules (tubulonephritis), or may be scattered throughout the kidneys, involving also the interstitial tissue (diffuse nephritis).

Glomerulonephritis is the type found in scarlatina, in streptococcus infections, and in poisoning by cantharidin. Bacteria, lodged in clumps, may be found in the capillaries occluding the lumen. The toxin of the organisms or the poison, *i.e.*, cantharidin, may injure the capillary wall, thus producing an occluding thrombus, and the loops thus occluded become greatly distended by a mass of hyaline material. The lumen of the capsule generally contains albuminous material consisting of blood, leucocytes and fibrin. These alterations interfere greatly with the permeability of the glomerules, and usually are associated with changes of an inflammatory char-

•

acter in the surrounding connective tissue of the capsule. The epithelium of the tubules later degenerates, and globules of neutral fat and cholesterin esters are found in the protoplasm, provoking a swollen appearance of the cells. Subsequently, the latter shrink in size and finally disappear, causing collapse of the tubule.

The presence of wandering cells in the connective tissue about the tubules indicates inflammatory reaction. The very marked disturbance of function is indicated by a great diminution in the quantity of urine, and its high concentration with the presence of blood, albumin and casts of various kinds. Edema of the subcutaneous tissue is very apt to be present.

When the lesions affect chiefly the tubules, as in poisoning by bichlorid of mercury, potassium bichromate, and similar agents, there is a very marked degeneration and necrosis of the cells lining the tubules. The ultimate result of destruction of epithelium of the tubules must depend upon its extent, since if it be partial, the tubule may be perfectly relined from the cells which remain, while if it be complete, the collapse of the tubule will lead shortly to destruction of the glomerules and the formation of a scar once occupied by the whole structure.<sup>4</sup> Edema is not so likely to appear in cases of tubulonephritis. In diffuse nephritis the interstitial tissue is affected, as well as the glomerules and tubules, and this part of the kidneys is edematous and infiltrated with round cells. The vessels are distended.

Symptoms may appear gradually or with great suddenness. When the disease is caused by bacteria, as in streptococcus infection, scarlet fever, influenza, diphtheria and typhoid fever, the symptoms are very likely to occur late in the course of the disease or even during convalescence. Most usually the first indication of nephritis is diminution in the quantity of urine, with change from the normal amber or pale straw color to a deep yellow or reddish brown. Coincident with the urinary change may be noted fretfulness, loss of appetite and a change in the general appearance of the patient difficult to describe, but which immediately attracts the attention of the observant physician. In some instances the first evidence of the disease is the sudden appearance of edema of the subcutaneous tissues around the orbits or face, arms and

legs, with mental dullness and headache. Edema in heart disease usually first appears in the ankles toward evening, while in acute parenchymatous nephritis the dropsy first appears under the eyes in the morning.

The specific gravity of the urine is usually 1020 or higher, the color dark yellow, or even bloody, and the reaction usually hyperacid. Albumin is almost invariably present in considerable quantity, and in a few instances the urine becomes almost solid upon boiling. Hyaline, granular, fatty and epithelial casts are present, and at times blood-casts appear. Renal epithelium is present in considerable quantity, particularly in tubulonephritis. Even though no macroscopic evidence of blood be present, erythrocytes are almost always found in the sediment, some well preserved, others in shadow form or crenated. There is little or no increase in the number of leucocytes in the urine. The quantity of urine voided in twenty-four hours may be as small as 10 or 12 ounces (295.7 to 355 mls). In severe cases total suppression may occur and persist for days.

The edema, at first, is likely to be limited to the orbital region or the face and hands, and is occasionally unyielding, so that pitting on pressure is slight and sometimes entirely absent. The facial edema smoothes out lines of expression, giving the patient an appearance of apathy, which is sometimes belied by the alert mentality. Very often, however, there is actual mental dullness and drowsiness. Later the edema may increase, involving the eyes, back, scrotum, abdominal wall and anterior thoracic wall, and when such is the case the cause is likely to be cardiac, as well as renal. Effusions in the serous cavities may occur, and edema of the lungs or glottis may cause death. As a rule, however, the edema is not so extreme nor so extensive as in the chronic parenchymatous cases. When the epithelium of the tubules has suffered damage almost to the exclusion of the glomerules, as is the case in acute bichlorid of mercury poisoning, edema is very slight and often entirely absent. In a case recently reported by the authors<sup>5</sup> there was complete anuria for four days without edema and without evidence of uremia.

Headache is often a very troublesome symptom, and is due probably to toxemia or edema of the cerebral meninges.



Nausea and vomiting, usually toxic in origin, are common, and in some instances even water is not retained in the stomach. Constipation is more commonly observed than diarrhea. Retinitis, with or without hemorrhages, is present in some cases.

Pericarditis is a rather frequent complication in cases of microbic origin, and may cause a fatal termination. Lobular pneumonia is sometimes found. Lobar pneumonia as a complication is rare.

The occurrence of a purpuric eruption indicates a severe infection, probably streptococcal, and has a very grave prognostic significance.

Uremia may occur during the course of the disease, and may cause death in convulsions and coma. Uremic manifestations are usually seen in terminal stages of chronic forms of the disease.

Probably a considerable number of cases of acute nephritis make a complete recovery. One is, however, not justified in recording a case as cured until the patient has been symptom-free for months, and repeated urinalyses have demonstrated the persistent absence of the disease, because there is a strong tendency to a transition from an acute to a chronic process, during which transition there is likely to be a very great amelioration of all the symptoms and signs of the disease.

Since acute nephritis is so likely to occur in those suffering from acute infectious diseases, notably scarlet fever, influenza, diphtheria and typhoid fever, it is advisable to regard these patients as potential nephritics, and throughout the course of the infection to attempt in every manner to protect the kidneys. As the bacteria and toxins generated in the infectious diseases are eliminated to a large extent through the kidneys, one should aim to dilute the urine so as to render them as little irritating as possible. This may be accomplished by the administration of water by mouth, 2 to 3 pints (1 to 1.5 l.) *per diem* being given, in addition to the liquids ingested as foods, *i.e.*, milk. A record of the quantity of liquid ingested and the quantity of urine excreted daily should be kept. The amount excreted by an average adult should be 50 to 60 ounces (1500 to 1800 mls), and is usually from 2 to 8 ounces (60 to 240 mls) less than the quantity ingested. If

diarrhea, vomiting or profuse sweating occurs, the difference will be greater. If, in the absence of any adequate cause, the difference is very great, it indicates an overtaking of the kidneys and circulatory apparatus, which is, of course, harmful. It is signally important to arrest polyuria, as it causes excessive renal work. If for any reason such as vomiting and certain types of delirium in which the patient cannot be aroused sufficiently to swallow, the water must be given per rectum, by the addition of sufficient glucose to make a 2 per cent. solution, the absorbability of the water will be increased and the tendency to rectal irritability lessened. The solution should be given either by the drop method or by continuous enteroclysis.

The apparatus necessary for the use of the drop method consists of a soft rubber rectal tube or catheter, a reservoir with a capacity of at least one quart, rubber tubing to connect the rectal tube with the reservoir, and a pinch cock. One pint (473.1 mls) of the dextrose solution at a temperature of 105° to 110° F. (40.5° to 43.3° C.) is placed in the reservoir and the diameter of the connecting tube constricted until the desired number of drops per minute escape from the rectal tube. The rate of flow in most cases should be 30 drops (1.9 mls) per minute. A stoppered bottle containing water at 105° to 110° F. (40.5° to 43.3° C.) is placed in the reservoir to maintain the temperature of the surrounding liquid, and changed as often as necessary to keep the temperature at the desired degree. The rectal tube is inserted about 4 inches (10.1 cm.) into the rectum, and may be retained in place by a bandage or strips of zinc oxid plaster.

The same apparatus may be used for continuous enteroclysis, the technic of which differs from the drop method in that the reservoir is placed but 2 or 3 inches (5.0 to 7.6 cm.) above the level of the patient's buttocks, and the connecting tube is not constricted. Hydrostatic pressure regulates the quantity of water in the rectum, increasing pressure in which will force some of the fluid back into the reservoir, while decreasing pressure allows more of the solution to enter the rectum. It is not necessary, in this method, to resort to measures to keep the water in the reservoir warm.

In view of the fact that the kidneys are irritated by the

toxins of disease, the administration of certain drugs, which in toxic doses produces a nephritis, should be avoided. Of such drugs, mercury, turpentine, salicylic acid, copaiba, potassium chlorate, and carbolic acid are striking examples. The skin should be kept in good condition by warm baths. Rubs with alcohol of greater strength than 50 per cent. should not be employed, because of the likelihood of removing the natural oil and impairing the efficiency of the skin. Hydrotherapy may be necessary to combat high fever, and for that purpose cold sponge baths are better than cold plunge baths or cold packs.

A daily examination of the urine is important, in order to detect abnormalities such as hyperacidity and concentration, the early recognition and counteraction of which protects the kidney.

If the disease be the result of the taking of drugs or the inhalation of turpentine (as may result from sleeping in a freshly painted room), these causative factors should be removed. The patient should be confined to bed and kept warm, and, if necessary, should sleep between blankets and wear flannel night clothes. The temperature of the room should be maintained at 70° F. (21.1° C.), but care must be taken to see to it that the air is fresh. Excitement and noise in the sick room should be avoided, as it is important to rest the mind as well as the body. For this reason, also, the patient should not be allowed to transact business. Usually the maintenance of mental rest is the most difficult task, and frequently the co-operative efforts of relatives and friends must be enlisted.

The diet is of great importance, and frequently becomes a very difficult problem. A cardinal principle in the treatment of acute nephritis is to lighten the work of the kidneys as much as possible. The chief indication is to avoid foods which may irritate the kidneys or increase the work of those organs. In severer cases no food of any kind need be given for several days, especially if nausea and vomiting are troublesome. Later skimmed milk is given, either alone or combined with lime-water. The addition of the latter is often useful when nausea follows the taking of milk. Six ounces (180 mls) of skimmed milk are given every three hours

between 7 A.M. and 10 P.M., and once during the night, if the patient is awake. As this supplies but 300 or 400 calories a day, the skimmed milk diet must not be continued more than a few days or a week, at the end of which time whole milk is substituted for a few days, thus raising the number of calories to approximately 600 per day. As improvement in the kidneys goes on, the caloric value of daily food consumption is increased by the addition of cream, sugar and other carbohydrates, such as crackers, zwieback, toast and rice. Meats should be avoided, because the acutely inflamed kidneys eliminate with difficulty the products of nitrogen metabolism. Creatinin is eliminated with difficulty by the diseased kidneys, and as this substance is plentiful in meat extracts and broths, these should be prohibited. Sodium chlorid should be strictly interdicted, if there is edema; otherwise, it may be used sparingly. Oranges, lemons and grapefruit may be allowed. It is most important to avoid intestinal toxemia, because the added burden thrown upon the functionally impaired kidneys may be sufficient to induce uremia.

In cases in which the kidneys are unable to eliminate water, as in glomerulonephritis, edema will be observed. In these cases the intake of fluid must be limited, otherwise the dropsy will be increased, and a burden thrown upon the circulation that may provoke dilatation of the heart, thus adding very materially to the gravity of the case. When there is no edema the amount of liquid need not be restricted, and in these cases the patient should be encouraged to drink freely of water. There are many waters recommended in Bright's disease, but it is very doubtful if their value is any greater than that of plain, pure water.

In the early stages of the disease elimination through the bowels and skin is of the utmost importance. The bowels should be made to move four or five times daily for the first two or three days, and for this purpose sodium sulphate or sodium phosphate, or both, should be used. If, because of nausea, the salines cannot be used, elaterin or compound jalap powder should be employed.

The skin is a very important organ of elimination, and should be kept warm and in good condition by a daily warm bath with castile soap and water. Free sweating should be



induced once daily, preferably by hot packs or electric light baths. If the patient is not very ill he may take a hot tub bath at a temperature of from 105° to 108° F. (40.5° to 42.2° C.) for from five to eight minutes, and then immediately return to bed between hot blankets, with hot-water bottles.

A hot wet pack is a very efficient method of inducing free sweating, and is given as follows: The top bed covers are replaced with a blanket, a rubber sheet or oilcloth, or several thicknesses of paper are placed over the mattress to keep it dry, and over this is spread a blanket. The patient is first wrapped in a dry sheet, and then covered with the top blanket, until the two wet blankets are ready. These are prepared by soaking them in water, the temperature of which is about 150° F. (65.5° C.), leaving one corner of the upper and lower edge of each blanket out of the water. To facilitate wringing, these corners should be diagonally opposite each other. Having wrung the blankets very dry, one is passed under the patient, and the other one placed over him, the ends being carried under and around the arms and adjusted so as to bring the corners over the shoulders; the legs are then enclosed, and the ends of the hot wet blanket upon which the patient is lying wrapped about him. He is then covered with a dry blanket, the ends of which are tucked under the shoulders, sides and feet. The left edge of the under dry blanket is then brought over and tucked under the right side, then the right edge of the blanket is brought over and tucked under the left side. A towel is placed between the blankets and the patient's neck and chin. Hot-water bottles are placed along the sides and at the feet of the patient, and then the oilcloth or rubber sheet is brought over the patient, and over this is spread the bed clothes. Finally, an ice cap is placed upon the head.

Sweating begins promptly in most cases, and may be continued from ten to thirty minutes, depending upon the state of the circulation. The pulse is studied at the temporal artery, and if irregularity occurs, the bath must be discontinued. While in the bath a glass of hot lemonade, hot water, or Vichy should be given.

Care must be taken not to burn the patient. The common cause of burns are: Wringing out the blankets in water that



is too hot, or not wringing them dry enough; placing the hot-water bags next the wet blankets and thus causing steam. It is also very important to prevent the entrance of air under the blankets while the patient is lying in the pack, as this would cause chilling of the skin and induce an increase of the renal congestion. For the same reason chilling must be avoided when the patient is removed from the pack, by taking the precaution to remove the wet blankets under cover of the dry blanket.

In homes lighted by electricity an electric light bath may be employed to induce sweating. The bed is protected by a rubber sheet, oilcloth or several layers of paper, and over it is spread a blanket, the right edge of which is spread over the patient, and tucked under his left side. The left edge is tucked under the right side, and a towel is placed between the blanket and the patient's neck and chin. A frame of iron rods or of barrel hoops is arranged over the bed from the patient's neck to the feet, and from these hoops is suspended a board containing six to eight incandescent electric light bulbs. Care must be taken to avoid defective insulation of the wires. The frame is then covered with a blanket, over which is spread a rubber sheet, oilcloth, or several layers of paper, and over these another blanket. Currents of air from the outside must not be allowed to enter the improvised cabinet.

A hot-air bath may be used to induce sweating. A frame such as described for the electric bath is placed over the patient, and hot air from an alcohol or kerosene lamp resting upon the floor is conducted within the cabinet through a metal pipe. If the patient is able to leave the bed a cabinet bath may be employed. The cabinet consists of a square enclosure made of moisture-proof material, so arranged that the top fits snugly about the neck of the subject, who enters the cabinet nude, and sits on a chair with a solid wooden seat. An alcohol lamp under the chair supplies the heat. Cold compresses or an ice-bag should be placed on the head. Sweating, which, as a rule, begins quite promptly and is profuse, should not be allowed to continue too long. The bath should be discontinued at the end of fifteen or twenty minutes, and immediately upon leaving the cabinet the patient's body

should be quickly sponged with warm water and dried, the patient getting into bed immediately thereafter. Chilling can occur very quickly after leaving the cabinet, in which the temperature is 160° F. (71.1° C.) or higher. The room in which the bath is taken must, therefore, be warm, and the cabinet placed not only in the same room as the bed, but also as near the bed as is conveniently possible.

The induction of diaphoresis by the use of pilocarpin may be fraught with so much danger that only in those rare instances where sweating cannot be induced by one of the physical measures just indicated, is its use justified. It is a distinct cardiac depressant, and in acute nephritis a great strain is suddenly thrown upon the myocardium by the action of this drug. It is, therefore, bad practice to administer a drug which reduces the ability of the heart to do its work. Furthermore, pilocarpin not only stimulates the sweat glands to action, but also may induce such a profuse bronchial secretion as seriously to embarrass respiration, or even cause death. Finally, the diaphoresis produced by pilocarpin cannot be checked at will, as is the case with that produced by external means.

*There are no drugs that have a specific influence in acute nephritis.* Cantharides, copaiba, cubebs, turpentine, and gin do positive harm. The best diuretics are milk and water. Lemonade may take the place of some of the water. The addition of a teaspoonful (4 mils) of cream of tartar to the pint of lemonade increases the diuretic action, and has a somewhat laxative effect. The cream of tartar should first be dissolved in hot water, as it is not very soluble in cold water. Sodium bicarbonate by mouth or per rectum is often beneficial when toxemia is marked. When the heart is weak, especially if there is a falling blood-pressure, digitalis is indicated. The infusion, freshly made from assayed leaves, is the best preparation to use. The dose should be 2 to 4 drams (7.5 to 15 mils) every three or four hours. Sodium theobromin salicylate (diuretin) in doses of 10 grains (0.65 Gm.) and caffeine, are also valuable when the heart shows signs of failing.

When edema of the lungs occurs, venesection is often of great service. It is very easily accomplished as follows: The skin over the median basilic vein, preferably of the left arm,

is washed with soap and water, followed by alcohol, and then painted with 10 per cent. tincture of iodine. The arm is tightly constricted above, but complete constriction of the brachial artery should be avoided, because if some arterial blood is pumped into the arm the pressure in the constricted vein is higher, and bleeding occurs more readily. When the vein is well dilated a sharp-pointed curved bistoury is plunged through the skin into the lumen of the vein, which is incised by carrying the bistoury outward to the surface.

The quantity of blood withdrawn varies with the individual patient. As a rule, in adults from 8 to 30 ounces (240 to 900 mls) are withdrawn. The bleeding is stopped if faintness or nausea, not due to psychic shock, occurs.

Dry cupping over the lumbar regions is sometimes used. Its value lies in the relief of pain in these regions. It has no influence on the course of the disease in the kidneys. The technic is as follows: A swab wet with alcohol is ignited and held in a small thick glass for a few seconds. Then quickly place the glass over the region to be cupped. Apply in this manner as many glasses as necessary to cover the area to be cupped. Heating the air in the glass causes the air to expand. As cooling takes place a partial vacuum is formed, to fill which the skin is drawn into the glass. The glasses should be removed when the skin within becomes a deep red. If they are allowed to remain too long, ecchymosis will result. In removing, insert a finger under the rim, otherwise pain will be caused whenever much tissue has been drawn into the glass.

The application of heat, *e.g.*, a hot-water bottle, to the lumbar region will answer the purpose as well as does the cupping.

### CHRONIC PARENCHYMATOUS NEPHRITIS.

This, a chronic inflammation of the kidneys, of which the epithelial degeneration is the most prominent feature, is not always easily differentiated from chronic interstitial nephritis. In many instances it seems to develop from the acute form. Just how often this happens it is impossible to say, as acute nephritides, which become symptom-free, pass out of obser-

vation; and in the study of the chronic cases dependable histories are often impossible to obtain.

The disease develops so insidiously and advances so imperceptibly that its inception escapes notice, and for this reason a cause is often difficult to find. In general, it may be stated that the same factors which induce acute nephritis are capable of causing the chronic type. The difference in effect depends upon the amount of injury, and the length of time in which the injurious agent is effective. Thus, infection with virulent streptococci would, in all probability, result in an acute inflammation; while infection with less virulent organisms might give rise to a low grade inflammation, tending toward chronicity. Recurring mild infection is a common cause of latent chronic parenchymatous nephritis.

The types of streptococci provocative of chronic endarteritis and arthritis are capable also of producing a chronic diffuse renal inflammation. The most common foci from which these organisms are distributed are the teeth, tonsils, accessory sinuses, mastoid, middle ear, prostate, seminal vesicles, gall-bladder, and appendix; sometimes they are derived from a salpingitis.

Among the specific causes that are usually given for chronic parenchymatous nephritis, when it is not secondary to acute nephritis, are various chronic constitutional diseases, especially those which lead to anemia and cachexia. In this connection chronic tuberculosis must be mentioned. It has been estimated by some that 25 per cent. of the cases of chronic parenchymatous nephritis are in association with tuberculosis. The disease is very commonly found in children affected with hereditary syphilis. Estivo-autumnal malaria seems to play a rôle in etiology. Chronic endocarditis is often assigned as a cause of chronic renal diseases; it is probable, however, that there is no direct causal relationship, but, rather, that the factor accountable for the endocarditis also excites the renal inflammation.

In a study of the etiology, the occupation of the patient must be investigated, because of the fact that exposure over a long period of time to mercury, turpentine, carbolic acid, tar, naphthol, glycerine, oxalic acid and sulphuric acid may cause the disease in question. Exposure to great heat or cold,

as in the case of glass blowers, iron workers, stokers confined to hot engine rooms, is a predisposing factor.

Almost always the kidneys are found enlarged. It is to be remembered that the process is diffuse, and it is owing to the variable degree of implication of the different structural units of the kidney that various types have been described.

The leading type is the large white kidney, in which the renal epithelium is chiefly affected. It is light gray in color, with prominent stellate veins. The capsule strips easily and the renal substance is soft. On section the cortex is seen to be swollen, and may be as wide as the medulla, instead of about one-third of its width, the normal proportion. The color on section is grayish, and, in some instances, minute reddish dots betray the areas in which hemorrhages have occurred. The pyramids are dark in color.

Microscopically, the cells of the tubules are swollen, granular, and fatty. In some the nucleus has disappeared. The lumen of the tubule, in some places, is diminished by the swollen cells; in other parts it is greater than normal, because the living cells have disappeared. Casts of various kinds, erythrocytes and degenerated epithelium, are found in the tubules. Some of the Malpighian bodies are enlarged, and the epithelium of the glomerules and Bowman's capsule are in a state of fatty degeneration. The connective tissue between the tubules is edematous, and areas of round cell infiltration are observed. Small hemorrhages in the connective tissue are sometimes seen.

The disease begins insidiously and progresses slowly. The *symptoms* are likely to be vague, and not in the least suggestive of serious renal disease. The patient probably consults the physician because of malaise, poor appetite, disinclination to do his usual work, and fatigue without adequate cause. Weakness and headache may be the first symptoms noted. The importance of investigating thoroughly the cause of all such symptoms cannot be urged too strongly, because the diagnosis thus may be established long before the obvious symptoms and signs of nephritis appear.

The most characteristic evidence of the disease is obtained by an examination of the urine which, as a rule, is reduced to



one-third the normal daily quantity. The specific gravity is normal, or even increased during most of the course of the disease, but later it may become extremely low when renal insufficiency becomes very great, on account of failure of the renal cells to excrete urinary solids. A low specific gravity of a urine, with diminution of the quantity, is an unfavorable sign. In the absence of infection of the bladder, and when uninfluenced by medication, the urine is acid; sometimes decidedly so. Albumin is present in large amounts, 0.5 to 2 per cent. by weight and 25 per cent. or more by volume. The urine may be turbid because of the presence of urates. Hyaline and granular casts are present in abundance, and epithelial and waxy casts may also be found. During an acute exacerbation erythrocytes and blood-casts appear. Renal epithelium in all stages of degeneration is found in large quantities.

There is probably no other type of nephritis in which edema is present to such a degree, although early in the course of the disease it is absent. As a rule, it is observed first simply as a puffiness about the eyes, perhaps even limited to the lower eyelids. As the disease progresses, the entire face becomes swollen and the lines of expression are obliterated, and finally the subcutaneous tissue of the entire body becomes greatly edematous. The loose areolar tissue of the scrotum and penis may become so distended with fluid that much distress is caused and urination interfered with to a marked degree. Large quantities of fluid may accumulate in the peritoneal, pleural, and pericardial cavities, greatly embarrassing the action of the heart and lungs. Edema of the mucous membrane of the gastrointestinal tract prevents the digestion and absorption of food, as a result of which weakness and anemia occur.

The anemia is seldom severe, the erythrocytes are rarely less than 3,000,000 per cmm., with a commensurate loss of hemoglobin. The pallor of the skin is out of all proportion to the intensity of the anemia, giving the patient a very characteristic wax-like appearance. This pallor is due to a marked hydremia as well as to anemia. The majority of cases terminate fatally in one or two years. In some instances a rather unexpected change, apparently for the better, takes

place. The edema vanishes, the urine increases in amount, the albuminuria diminishes to a very small quantity, and all casts, except a few hyaline and granular, disappear. This usually indicates the transition from chronic nephritis, without induration, to chronic nephritis with induration, in which event the course of the disease is somewhat lengthened, but eventually death occurs from chronic diffuse nephritis.

Unfortunately no remedies exist that will *cure* chronic parenchymatous nephritis, but this does not mean that the mortality rate in this type of renal disease is 100 per cent. A few cases are reported in which the inflammation was arrested and regeneration occurred to such an extent that the patient became entirely well. When the disease is due to recurrent infection from a focus elsewhere in the body, and the original focus is removed, the best opportunity is afforded for complete recovery. In the vast majority of cases, however, the best we can hope to accomplish is the prolongation of life, with as much freedom from distressing symptoms as possible. It must be realized that the ability of the kidneys to properly functionate is diminished, and that, therefore, the patient must live within his renal efficiency; the demands made upon the kidneys must be greatly reduced. This is done by regulating the daily activities of the patient and arranging his diet so that the end-products of its metabolism may be eliminated with ease, and are non-irritant in their passage. It is of the utmost importance to avoid intestinal toxemia.

Obviously, rest is an important part of the treatment, but in a disease that lasts for many months, strict confinement to bed is not only ill-advised, but may be actually injurious by its monotony and the consequent depression of the entire nervous system. On the other hand, experience teaches that rest in the horizontal position relieves the kidneys, as shown by the diminished quantity of albumin in the urine voided upon arising in the morning, as compared with the quantity found in the specimen passed upon retiring. The aim should be, therefore, to obtain as much rest in recumbency as possible. In patients with little or no edema, and not more than a moderate amount of albuminuria, ten to twelve consecutive hours in bed should be prescribed; and in the early afternoon

the patient should spend one or two hours in recumbency. Physical exertion that would be very moderate for one in health is to be regarded as excessive in chronic parenchymatous nephritis. Ascending steps, walking long distances on level ground, and going up hill should be reduced to a minimum, the patient being instructed to use a conveyance to get about from place to place. In this way it is possible for considerable business to be transacted with a minimum of physical fatigue. Standing for long periods must be avoided. When the urine becomes scanty and edema increases, absolute confinement to bed becomes necessary, and must be continued until the edema lessens and the urine increases in amount. Acute exacerbations are common, and must be treated as an acute nephritis.

Prolonged exposure to cold and wet should be avoided. When possible the patient should leave a cold, wet climate, and live in a locality where it is dry and warm. The warmth dilates the peripheral vessels and increases the elimination through the skin. In such a climate the patient is enabled to be out-of-doors, which is of distinct advantage. Suitable winter climate may be found in southern Texas, southern California, and low altitudes in Arizona and New Mexico. Those unable to secure the advantages of suitable climate must dress warmly. Flannels should be worn next the skin. On cold stormy days the invalid must stay indoors.

In order to keep the skin active, a warm cleansing bath should be taken daily. Cold baths of all kinds must be avoided. Sea bathing, because of the low temperature of the water and the exertion necessary, must be strictly forbidden. Sweating should be induced by means of hot baths, as described on page 586. The frequency of the sweat baths must depend upon the condition of the patient. Weakness follows their use, so that they should be employed, generally speaking, but once or twice a week, when elimination is not entirely satisfactory. Turkish baths may be substituted for the sweat baths. In acute exacerbations sweat baths must be used daily as in acute nephritis.

Diet is one of the essential parts of treatment. It should be arranged so that the proteins are just sufficient to supply the needs of the body, remembering that muscular activity

has been greatly reduced. The bulk of the food should be selected from the carbohydrates and fats. It is impossible to give a standard of protein diet that will be applicable to all cases, or even to the same case throughout the entire course, since the functional power of the kidneys and gastrointestinal tract varies in different phases of the disease, and also because the needs of the organs vary from time to time; the quantity required when the patient remains absolutely quiet in bed will be less than when he is going about.

When the urine is scanty and contains a large amount of albumin, when edema is very much in evidence and symptoms of uremia are present, the diet is restricted to 8 ounces (240 mls) of skimmed milk, taken at intervals of four hours between 8 A.M. and 8 P.M., and when anasarca is present, absolutely no other fluids than this are allowed. As improvement is made, crackers or zwieback are permitted with the milk, and gradually the diet is increased by the addition of eggs, cooked cereals, toast, butter and vegetables, such as rice, potatoes, barley, hominy, spinach, green beans and peas.

It is estimated that a minimum of 40 to 50 Gms. (1.28 to 1.6 ozs.) of proteins are required by the body daily, and if not supplied by the food the proteins of the body will be utilized, causing emaciation. In the treatment of nephritis the proteins may be greatly restricted for a time, but as soon as possible the minimum physiological amount of proteins must be supplied. This may be done by giving increased quantities of milk. Later it may be given in other forms, such as eggs, meats and fish, but the daily quantity consumed should be kept under 50 Gms. (1.6 oz.). The following list will give the quantity of raw foodstuffs that would yield 50 Gms. (1.6 oz.) of protein:<sup>6</sup>

Hen eggs .....	7		
Milk .....	1660	mls	1½ qts.
Lean beef .....	220	Gms.	7⅓ ozs.
Ox tongue .....	300	"	10 "
*Lean veal .....	260	"	8⅔ "
*Calf's liver .....	260	"	8⅔ "
Lean mutton .....	250	"	8⅓ "
*Lean pork .....	240	"	8 "

---

\* These should be excluded because of their indigestibility.

Very fat bacon .....	3000	Gms.	100	ozs.
Chicken breast .....	250	"	8 $\frac{2}{3}$	"
Pigeon .....	210	"	7	"
*Goose .....	320	"	10 $\frac{2}{3}$	"
*Rabbit .....	200	"	6 $\frac{2}{3}$	"
*Venison .....	230	"	7 $\frac{2}{3}$	"
Trout .....	260	"	8 $\frac{2}{3}$	"
Pike .....	260	"	8 $\frac{2}{3}$	"
*Herring .....	260	"	8 $\frac{2}{3}$	"
*Salmon .....	230	"	7 $\frac{2}{3}$	"
*Lobster .....	300	"	10	"
Oysters .....	800	"	26	"

## COOKED BOILED MEATS.

Lean beef .....	150	Gms.	5	ozs.
*Lean veal .....	190	"	6 $\frac{1}{3}$	"
Lean mutton .....	160	"	5 $\frac{1}{3}$	"
*Lean pork .....	175	"	5 $\frac{1}{2}$	"
Chicken breast .....	160	"	5 $\frac{1}{3}$	"
*Salmon .....	210	"	7 $\frac{1}{3}$	"

## BAKED OR BROILED MEATS.

Roast beef .....	200	Gms.	6 $\frac{2}{3}$	ozs.
Beef steak .....	200	"	6 $\frac{2}{3}$	"
Stewed beef .....	160	"	5 $\frac{1}{3}$	"
*Roast veal .....	200	"	6 $\frac{2}{3}$	"
Lamb chop .....	220	"	7 $\frac{1}{3}$	"

An adult of average weight will require about 2200 calories daily, if at rest, and about 3000 if working. If the patient be allowed the minimum of protein, 50 Gms. (1.6 oz.) equivalent to 200 calories, the remaining calories must be supplied from the fats and carbohydrates. It is a matter of indifference whether the white or the dark meats are allowed, the important thing being not to permit more than will supply the protein needs of the body. The same is also true of eggs. Smoked and salt meats, and fish, condiments and meat extractives, as beef tea, etc., should be avoided. Salt should be restricted, and when edema is present no salt whatever is permissible. The amount of salt consumed by the average individual is much in excess of the needs of the body, which is about 1 or 2 Gms. (15.4 to 30.8 grs.); when edema is absent this quantity may be allowed.

---

\* These should be excluded because of their indigestibility.



The moderate use of tea and coffee may be permitted, unless sleep is affected. Alcohol is harmful. As nicotin is a poison to nerve tissue and to the heart, tobacco should be avoided by the patient.

Diuretics which act chiefly on the glomeruli, stimulating them to increased activity, and which do not irritate the epithelium of uriniferous tubules, should be used, not routinely, but as necessary. The value of water, milk and lemonade as diuretics should be kept in mind. Potassium bitartrate (cream of tartar), 15 to 30 grains (0.9 to 1.9 Gms.) may be added to the lemonade. Infusion of digitalis, 1 to 4 drams (4 to 16 mls), every four hours is often of great value. Potassium citrate, 15 to 30 grains (0.9 to 1.9 Gms.), sometimes seems to be of assistance.

In some cases, late in the course of the disease, the heart, hypertrophied in its effort to meet the new demands thrown upon it, dilates, causing an increase in the amount of edema, and it is especially in these instances that the effusions into the serous cavities take place. It then becomes necessary to prohibit the use of salt in the food, and an attempt to carry off the fluid by copious bowel movements must be made. The use of diuretics, preferably of the digitalis group, is demanded. If the effusion in the serous cavities increases in amount, and causes symptoms, or gives evidence by physical signs of embarrassed action of the heart and lungs, the fluid must be removed by aspiration.

In aspiration of the pleural cavity the skin of the area to be tapped must be washed with soap and water and dried; then tincture of iodine is applied and allowed to remain for a few minutes before being removed with alcohol. The site selected is the seventh interspace between the posterior axillary line and the scapula, or immediately below the angle of the scapula, it having been determined beforehand, by physical examination, that the lung is pushed away from that locality. After the apparatus has been sterilized, it is tested to see that it works properly. It consists of a bottle, a pump for exhausting the air from the bottle, and a trocar and canula or needle, with rubber tubing to connect the canula and the pump with the bottle. The trocar and canula, or

needle, should be at least 6 inches (15.2 cm.) long with a diameter of not more than  $\frac{1}{8}$  of an inch (3.1 mm.). The entrance of air into the bottle through the needle is prevented by clipping the connecting tube with a hemostat. The air is then exhausted from the bottle. The needle is held in the hand in such a manner that the proximal end is in contact with the center of the palm, while the index finger rests on the shaft about 3 inches (7.6 cm.) back from the point. With firm pressure the needle is pushed into the interspace until the tip is within the pleural cavity. The hemostat is removed from the tube and the liquid allowed to flow slowly into the bottle. It is not necessary to draw off the entire quantity of the fluid, two-thirds of the quantity being enough. The remainder is usually quickly absorbed. Faintness and vertigo occurring during the tapping, unless due to psychic shock, indicate immediate withdrawal of the needle. If severe coughing begins after considerable of the fluid has been withdrawn, the operation must be discontinued. A collodion dressing is sufficient to close the wound made by the needle.

The accumulation of a large quantity of fluid in the peritoneal cavity is likely to cause passive congestion of the kidneys by the pressure of the fluid on the renal veins. This would be indicated by a diminishing quantity of urine containing microscopic, and, rarely, macroscopic blood. The upward pressure exerted upon the diaphragm is likely to interfere seriously with the action of the heart and lungs. If the fluid does not diminish in a short time as a result of measures directed towards the relief of general edema, the fluid must be relieved by aspiration, as has been described, or by a trocar and canula, as follows: The skin over the abdomen is prepared as was advised for thoracentesis. The sterile trocar and canula are held in the hand, so that the proximal end of the instrument is in the palm of the hand and tip of the index finger 3 or 4 inches (7.6 or 10.1 cm.) back from the point of the instrument, which is plunged through the abdominal wall in the midline, equidistant from the umbilicus and pubes. A small incision through the skin may be made at the site selected before inserting the trocar. The urinary bladder should be empty.

The fluid should be withdrawn slowly, and as its level falls, a many-tailed binder should be applied so as to prevent too great reduction in intra-abdominal pressure. Death has followed the too rapid evacuation of a peritoneal effusion through a large canula.

In some instances after removal of fluid there is no recurrence, but not infrequently there is a reaccumulation, and repeated tapings are necessary, as the transudate reaccumulates from time to time.

### CHRONIC INTERSTITIAL NEPHRITIS.

Chronic interstitial nephritis is characterized by a great increase in the connective tissue of the kidney with a lesser degree of fibrosis affecting the glomerules and the cells of the tubules. There is always an associated cardiac hypertrophy, and a variable degree of arteriosclerosis.

In the great majority of cases no causative factor can be assigned. The disease is insidious, of long duration, and so many causal factors may be at work, that it is, as a rule, impossible to select one and ascribe to it the production of the disease.

Undoubtedly the disease follows recurring acute nephritis. In the course of an acute infectious disease a more or less severe nephritis develops with or without dropsy, runs a favorable course, but leaves behind a slight intermittent albuminuria which is overlooked or neglected, until after a time unmistakable signs of chronic nephritis make their appearance. The disease may have its origin also in the nephritis of pregnancy.

Among the infectious diseases capable of initiating chronic nephritis should be mentioned influenza. It is very likely that recurring infection of the kidneys with an organism of low virulence from a focus in the tonsils, sinuses, or about the teeth, may account for a large number of cases of this disease.

A subdivision of chronic interstitial nephritis—arteriosclerotic kidney—is made by many writers. Such a subdivision has little value clinically, but is interesting only because it draws attention to the fact that the cause which produced the nephritis was also very active in producing vas-

cular and cardiac disease. There is still considerable discussion over the relationship of chronic interstitial nephritis and arteriosclerosis. Senator says the relations existing between induration of the kidneys and arteriosclerosis are of a triple character: (1) As a result of certain noxious agents arteriosclerosis may be the primary affection and bring about induration of the kidneys, forming the so-called arteriosclerotic induration of the kidney or contracted kidney. (2) Conversely, induration may result from chronic interstitial nephritis, and later, on account of the cardiac hypertrophy and associated increase in the arterial tension, cause a vascular sclerosis. (3) The two conditions may develop independently of one another from the same cause.

Alcohol is an important etiological factor. There is probably little action by alcohol as such on the renal tissue. The main influence is through its action on the gastrointestinal tract and liver, causing disorders of digestion and abnormalities of metabolism, as a result of which toxic substances are elaborated, which in their elimination through the kidney excite the inflammation. Alcohol produces cirrhosis of the liver, and, in association, this condition is found in almost every instance of induration of the kidneys. Excessive beer drinking over a long period of time causes interstitial nephritis, largely through the effect upon the circulatory apparatus of the kidneys.

Long-continued gastrointestinal indigestion, from any cause, may result in chronic renal disease. The same is true of certain metabolic disorders, such as gout and diabetes. Chronic intoxication with certain chemicals, notably lead, result in hardening of the kidneys.

The association of chronic interstitial nephritis and heart disease is very close. Usually the same cause produces both. Occasionally the disease of the kidney is secondary to the disease of the heart, and is the result of chronic passive congestion, just as cirrhosis of the liver (cardiac cirrhosis) is observed in many instances.

The kidneys in chronic interstitial nephritis are small and firm, with a rough surface, upon which may be found one or more cysts of varying size containing usually a pale, clear, straw-colored liquid. The capsule is thickened, strips with

some difficulty and adheres to the cortex so firmly in places that this substance is torn in the stripping of the capsule. On section the kidney tissue is resistant to the knife. Because of the diminution of the renal substance, the space in which the pelvis lies appears unusually large, and is often well filled with adipose tissue. The cortex is often much narrowed, often being only 2 to 3 mm. (0.06 to 0.09 inch) in width. The striæ are seen with difficulty. The pyramids appear larger than normal, but are not actually so. Owing to the atrophy of the cortex the bases of some of the pyramids are almost immediately under the capsule. The blood-vessels are rigid, and gape on section.

On microscopic study some of the glomeruli are enlarged, this change probably being of compensatory character. In others the capillary loops are so thick that they are impermeable to blood. The capsule about some of the glomeruli is very greatly thickened, and filled with a hyaline mass showing complete destruction.

Some of the tubules are enlarged through compensatory hypertrophy, others dilated as the result of constriction of their distal lumen, and still others are atrophied. The cells of some of the tubules present evidences of degeneration. The protoplasm is very granular, and contains hyalin and fat droplets.

The connective tissue outside the glomeruli, and between the tubules, is increased in amount. In its meshes the blood-vessels, obliterated glomeruli, and traces of tubules become very closely arranged, because of the disappearance of so many structures which were there before. There is more or less round cell infiltration.

In addition to the pathologic changes in the kidneys, hypertrophy, and sometimes, dilatation of the heart, chiefly of the left ventricle, are found. The heart muscle usually shows some evidence of degeneration. The blood-vessels are thickened and dilated, and their elasticity is diminished. Extreme arteriosclerosis and atheroma occur sometimes in association with chronic interstitial nephritis.

It is one of the unfortunate characteristics of this disease that for a long time after the pathologic changes have begun in the kidney, no symptoms occur. Were it otherwise, the



disease could be detected in a much earlier stage. By the time symptoms are noted, the disease is usually well advanced. The early symptoms are so vague that, unless a thorough study of the patient is made, the real cause of the symptoms may be overlooked. Among the earliest clinical manifestations are anorexia, loss of the power of concentration, and mental and physical fatigue after but moderate effort. Such symptoms are apt to be ascribed to overwork instead of to the true cause, namely, nephritis. Sometimes headache and momentary attacks of vertigo are the symptoms for which the physician is consulted. When a blood-pressure observation is made, using a sphygmomanometer, the systolic pressure is found to be 170 mm. or higher. In some instances in which slight symptoms are complained of, the blood-pressure has been found to be 250 mm.; in one instance 300 mm., and in another 320 mm. Patients may pay no attention to the slight symptoms, and the first intimation of serious trouble may be cerebral hemorrhage or acute uremia.

It is impossible to select a symptom-complex as representative of a disease where the symptomatology is so varied. Some cases will present symptoms that result from renal toxemia; others appear to present more the picture of a myocardial degeneration.

The urine is increased in amount, usually light in color, and of low specific gravity (1005 to 1012). There is very little sediment deposited, and on examination under the microscope a very few hyalin casts are the only abnormalities. Sometimes they are found only after a prolonged search. Albumin is present in very small quantity and is not constant. It is often absent in specimens voided upon arising in the morning, and sometimes for days and weeks at a time it may be absent from all specimens examined. It is, therefore, important to avoid the mistake of eliminating the possibility of interstitial nephritis upon the findings of but one urinalysis, in the absence of albuminuria.

Cardiac hypertrophy is constantly associated with indurated kidneys, even when no symptoms are manifest. At times the hypertrophy is very marked. The apex beat is displaced to the left and sometimes downward as well, and is usually well felt, unless dilatation has occurred. The mus-

cular element of the first sound is increased. The aortic second sound is accentuated, and sometimes is very loud and ringing. In some cases a systolic murmur, due to mitral insufficiency, is audible at the apex, and may or may not be transmitted to the left axilla. In the late stages pericardial friction rubs are sometimes heard. The superficial arteries are found to be much thickened, hard, and often tortuous. The radial pulse is hard and sometimes incompressible. Palpation of the pulse is not always a safe guide in studying the pressure. A pulse that is seemingly compressed with moderate ease may show on the blood-pressure instrument a reading of 200 mm., especially if the pulse pressure be increased. The reverse is also sometimes true.

Dyspnea on exertion is a common symptom somewhat late in the disease, and is dependent, in some instances, entirely on the condition of the heart. Toxic dyspnea, entirely independent of myocardial disease, is frequently observed. It may take the form of sudden attacks of suffocative feelings occurring during the night, awakening the patient from sleep, causing him to sit up in bed, or even to arise and sit in a chair at the window. Such an attack may last but a few minutes, and recur one or more times during the night; or, it may last for hours, during which the physical discomfort is associated with considerable mental suffering. Cheyne-Stokes breathing is an extremely common phenomenon late in the disease, and is of bad prognostic significance, as it indicates a pronounced degree of toxemia. Usually it is manifested a few months prior to death, but not uncommonly it may recur through one or more years. Often the patient is unconscious of this peculiar type of respiration and feels very comfortable. Some patients are very much distressed by its presence. The paroxysms of Cheyne-Stokes breathing are very likely to occur during the night.

Retinal changes may be detected in the great majority of instances of chronic interstitial nephritis, sometimes producing almost complete blindness, and sometimes not affecting the sight at all. The commoner changes in the retina are whitish or yellowish patches, hemorrhages and diffuse retinal opacity from edema. The arteries are thickened and the veins are enlarged.

Uremia, either acute or chronic, is very commonly observed in the terminal stages of the disease. The first evidence of acute uremia may be a clonic convulsion of great severity lasting a few minutes, after which consciousness is rapidly regained, followed for a short time by slight mental confusion or a somewhat dazed state; or, instead of regaining consciousness after the convulsion, the patient may become comatose and die. Sometimes the convulsions are preceded, for a few hours or a day or two, by suggestive premonitory disturbances such as severe headache, nausea, vomiting and restlessness.

In chronic uremia convulsions are less common. The chief symptoms may be drowsiness, or restlessness and insomnia, headache, nausea and vomiting after eating or drinking, or sometimes produced by the mere sight of food. Partial palsies of various muscle groups are often observed. Delirium may occur, and last for days.

It is evident that in dealing with a disease of the kidneys, so insidious and at the same time so serious, that the victim may be stricken with a fatal attack of uremia, any procedure that discovers the functional capacity of those organs is of the utmost importance. A number of tests are now being employed, no one alone being sufficient. As the function of the kidney is a complex one, carried on by various highly specialized units, it is not surprising to learn that various tests must be employed in the study of renal function. The tests that are of practical value are the phenolsulphonephthalein test, the nephritic diet test, and the estimation of the blood content of uric acid, urea, and creatinin.

The phenolsulphonephthalein test is very frequently and extensively used at the present time. It was devised by Rowntree and Geraghty,<sup>7</sup> and depends upon the injection into the tissues of a dyestuff which is eliminated rapidly by the normal kidneys, and can be easily estimated quantitatively in the urine. It is non-irritative, and does no harm to the kidneys.

The method of application is as follows: The patient is instructed to empty the bladder, and is given 6 to 8 ounces (180 to 240 mls) of water to drink. Then 1 mil (16 m.) of the dyestuff, which can be obtained in sterile ampoules, is

injected subcutaneously into the arm or other convenient part of the body. Normally ten minutes are required for the elimination of the substance to begin. One hour and ten minutes after the injection the bladder is emptied and the entire amount of urine voided is saved. One hour after this specimen is voided the bladder is again emptied, and the entire quantity saved. These specimens separately are measured, made strongly alkaline by the addition of 25 per cent. sodium hydroxid, and the quantities brought up to 1000 mls (1 qt.) by the addition of water. Comparison is made in a Duboscq or Hellige colorimeter with a standard consisting of 3 mg. ( $\frac{1}{20}$  gr.) of phenolsulphonephthalein in 1000 mls (1 qt.) of distilled water. Each ml of the dyestuff in the ampoule contains 6 mg. ( $\frac{1}{10}$  gr.). Since the volume of each urine sample is the same as that of the standard, the percentage elimination of phenolsulphonephthalein in each may be easily calculated as follows: Reading of urine: Reading of the standard :: 100: X. The amount of the drug eliminated normally is 40 to 60 per cent. for the first hour, and 20 to 25 per cent. during the second hour, or a total of 60 to 85 per cent. for the two hours.

The study of the quantity and of the specific gravity of day and night specimens, according to the method of Mosenthal,<sup>8</sup> sometimes referred to as the nephritic test diet, is of value. The patient is ordered to eat three full meals a day and to write down the approximate quantities; for example, one cup of coffee, two slices of toast, two tablespoonfuls of rice, in order to be certain that the diet for the day contains a sufficient quantity of the diuretic materials of ordinary food to make an adequate demand on the kidney. The urine must be collected punctually every two hours from 8 A.M. to 8 P.M., and a 12-hour specimen from 8 P.M. to 8 A.M. is also taken. No solid food or fluid of any kind must be taken between meals, and especial care must be observed that nothing of any kind is eaten or drunk during the night, and that the night specimen is completed before breakfast is touched. The volume in cubic centimeters and the specific gravity of each specimen are determined. After each meal there is normally an increase in the quantity of urine, with a wide variation of the specific gravity (about 10 points) and a small quantity of

urine, 400 mils (13.3 f $\bar{3}$ ), or less, between 8 P.M. and 8 A.M., with high specific gravity. In most normal individuals the urinary output is about 400 mils (13.3 f $\bar{3}$ ) less than the intake.

The kidney expresses its diminished power to functionate by a fixation of its concentration. The evidence of renal functional impairment consists of slight variation in the quantity and specific gravity of the two-hourly specimens and an increase over 400 mils (13.3 f $\bar{3}$ ) with little change in the specific gravity of the twelve-hour specimen.

The value of the study is increased if the chlorid and nitrogen excretion for the twenty-four hours is also estimated.

Much valuable information may be gained from a study of the concentration in the blood of uric acid, urea and creatinin. Uric acid is least readily, and creatinin most readily eliminated by the kidneys, urea occupying in this respect a position midway between the two. The studies of Chase and Myers<sup>9</sup> show that many early cases of nephritis, probably of the interstitial type, give blood-pictures in which the essential feature is the high uric acid content. The urea and creatinin are frequently normal, though sometimes appreciably increased. As the condition of the cases of this type becomes more severe, the retention of urea increases, until the picture is that of the preceding group. If, on the other hand, the case goes on to a fatal termination, the retention of uric acid and urea is followed by that of creatinin, the concentration of which may reach twenty times the normal. As a prognostic test the blood creatinin has been found of very great value.

The normal concentration of uric acid is 1 to 2 mg. ( $\frac{1}{64}$  to  $\frac{1}{32}$  gr.), urea 12 to 17 mg. ( $\frac{3}{16}$  to  $\frac{1}{4}$  gr.) and creatinin 1 to 2 mg. ( $\frac{1}{64}$  to  $\frac{1}{32}$  gr.) per 100 mils (3.3 f $\bar{3}$ ) of blood.

The technic of these tests, as employed by Myers, Fine and Lough,<sup>10</sup> is as follows:

**Uric Acid.** Ten mils ( $\frac{1}{3}$  f $\bar{3}$ ) of a mixture of blood and potassium citrate are pipetted into a casserole of about 375 mils (12.5 f $\bar{3}$ ) capacity and approximately 5 volumes of hundredth normal acetic acid added. The mixture is heated over a water bath, and finally brought to a boil over a free flame, stirring continuously. About 4 mils (64.8 *m.*) of



fairly thick alumina cream\* are added with continuous stirring for a few minutes. The sides of the dish are washed down from time to time with hot water, and the mixture then filtered through a hardened filter paper. The coagulum is returned to the casserole with about 150 mils (5 f $\bar{3}$ ) of hot water, heated and filtered through the same paper. The combined filtrates are evaporated to 1 or 2 mils (16 or 32.4 *m.*) (the material should be protein-free) and transferred to a 15 mils (0.5 f $\bar{3}$ ) conical centrifuge tube, washing the casserole with two or three small portions of hot water, but keeping the volume at or below 10 mils (2.7 f $\bar{3}$ ). About 15 drops (1 mil) of ammoniacal-silver-magnesium mixture† are now added, the tube shaken and placed in a cool place (refrigerator) for about fifteen minutes to allow for the precipitation of the purins. The tube is centrifuged, the supernatant liquid decanted off and allowed to rest in an inverted position for about five minutes. The tip of the tube is then wiped with filter paper and the ammonia allowed to volatilize, this change being facilitated with suction.

For the development of the color prepare a 100 mils (3.3 f $\bar{3}$ ) graduated cylinder for the standard and a 50 mils (1.6 f $\bar{3}$ ) cylinder for the unknown. Five mils of the uric acid standard‡ (5 mils = 1 mg. of uric acid) are pipetted into

---

\* Alumina Cream.—Prepare by precipitating an 8 per cent. solution of aluminum acetate in acetic acid with sodium bicarbonate, carefully washing with a large volume of distilled water by decantation several times, then filtering.

† Ammoniacal-silver-magnesium solution is prepared by mixing 70 mils (2.3 f $\bar{3}$ ) of 3 per cent. silver nitrate solution, 30 mils (1 f $\bar{3}$ ) of magnesia mixture and 100 mils (3.3 f $\bar{3}$ ) concentrated ammonia. Any turbidity which may develop is filtered off. Magnesia mixture is prepared by dissolving 35 Gms. (1.1 f $\bar{3}$ ) magnesium sulphate and 70 Gms. (2.2 f $\bar{3}$ ) ammonium chlorid in 280 mils (9.3 f $\bar{3}$ ) distilled water, and then adding 140 mils (4.6 f $\bar{3}$ ) concentrated ammonium hydroxid.

‡ The standard uric acid solution is prepared as follows: Dissolve 9 Gms. (138.9 grs.) pure crystallin hydrogen disodium phosphate and 1 Gm. (15.4 grs.) dihydrogen sodium phosphate in 200 to 300 mils (6.6 to 10 f $\bar{3}$ ) hot water. Filter and make up to 500 mils (16.6 f $\bar{3}$ ) with hot water. Pour this warm clear solution on 200 mg. (1000 mils) uric acid, suspended in a few cubic centimeters of water in a liter (quart) volumetric flask. Agitate until completely dissolved. Add at once exactly 1.4 mil (23.3 *m.*) glacial acetic acid. Make up to 1 liter (quart), mix and add 5 mils (1.3 f $\bar{3}$ ) chloroform. Five mils of this solution are equivalent to 1 mg. uric acid. The solution should be freshly prepared every two months.

the 100 mls (3.3 f $\bar{3}$ ) cylinder. To the standard are added 2 drops ( $\frac{1}{8}$  mil) of 5 per cent. potassium cyanid, 2 mls (32.4 *m.*) of Folin-Macallum reagent\*, 20 mls (5.3 f $\bar{3}$ ) of saturated (22 per cent.) sodium carbonate, and, in about one minute, water to the 100 mls (3.3 f $\bar{3}$ ) mark. To the precipitate in the centrifuge tube are added 1 or 2 drops ( $\frac{1}{16}$  or  $\frac{1}{8}$  mil) of the potassium cyanid, 2 mls (32.4 *m.*) of the Folin-Macallum reagent and 15 to 20 mls (4 to 5.3 f $\bar{3}$ ) of the standard sodium carbonate, depending on whether the color is subsequently diluted to 50 or 100 mls (1.6 or 3.3 f $\bar{3}$ ). After forty to sixty seconds dilute with water until the intensity of the color is similar to the standard, and then match in the Duboscq colorimeter. The prism of the standard may be set conveniently at the 10 mm. mark.

**Urea.** Into a test-tube (of such size that it will just slip into a 100 mls (3.3 f $\bar{3}$ ) cylinder) are introduced 1 mil (16 *m.*) of 10 per cent. urease solution, or about 0.1 Gm. (1.5 grs.) of the dry enzyme, and then 1 to 2 mls (16 to 32.4 *m.*) of water. Two mls of blood are now added with an Oswald-Folin pipette and the tube incubated in a beaker of water at 50° C. (122° F.) for about one-half hour. At the end of this time 3 to 4 drops (0.18 to 0.24 mls) of caprylic alcohol or 1 mil of amyl alcohol are added to prevent foaming in subsequent aëration. Into a 100 mls (3.3 f $\bar{3}$ ) graduated cylinder, without lip, are added 15 mls (0.5 f $\bar{3}$ ) of distilled water and 2 drops (0.12 mls) of 10 per cent. hydrochloric acid. This is now closed with a two-hole stopper having a glass tube passing nearly to the bottom of the graduate. The tube is sealed at the lower end, but contains a number of small holes to aid in the complete absorption of the ammonia. To the test-tube containing the digested blood an equal volume of saturated sodium carbonate is carefully added, the solution being allowed to run underneath the blood. The tube is now immediately inserted in a 100 mls (3.3 f $\bar{3}$ ) cylinder and a two-hole stopper is used containing one glass tube, which

---

\* Folin-Macallum reagent is prepared by boiling 100 Gms. (3.2 f $\bar{3}$ ) sodium tungstate, 20 mls (5.3 f $\bar{3}$ ) concentrated hydrochloric acid and 30 mls (1 f $\bar{3}$ ) 85 per cent. phosphoric acid in 750 mls (25 f $\bar{3}$ ) distilled water for two hours, preferably under a reflex condenser, and then making up to 1000 mls (33.3 f $\bar{3}$ ) with water.

passes nearly to the bottom of the tube. This is now connected on one side with a wash bottle containing dilute sulphuric acid, and on the other with the graduated cylinder containing the dilute acid for the absorption of the ammonia. The ammonia of the blood is now transferred to the cylinder containing the weak acid, by vigorous aëration for twenty to thirty minutes. At the end of this time the outfit is disconnected and nesslerization carried out in the graduated cylinder, dilution being made according to the amount of ammonia present.

Into a volumetric flask of 100 mils (3.3 f $\bar{3}$ ) capacity, if the Duboscq colorimeter is to be used, are pipetted 5 mils (1.3 f $\bar{3}$ ) of ammonium sulphate or ammonium chlorid solution containing 1 mg. (5 mils) of nitrogen.\* Fifty to 60 mils (1.6 to 2 f $\bar{3}$ ) of distilled water are added. Ten mils (2.7 f $\bar{3}$ ) of modified Nessler's solution† are now diluted about five times with distilled water, and of this 20 to 25 mils (5.3 to 6.7 f $\bar{3}$ ) added to the standard solution, which is then made up to the mark with water. At the same time 7 to 8 mils (1.8 to 2.1 f $\bar{3}$ ) of the freshly diluted Nessler's solution are added to the unknown solution and the volume made up to 25 mils (6.7 f $\bar{3}$ ) in the graduate, unless a high content of urea nitrogen is indicated, in which case more Nessler's solution, up to 25 mils (6.7 f $\bar{3}$ ), and a dilution of 33 $\frac{1}{3}$ , 50, 100 mils, 1.1, 1.6, 3.3 f $\bar{3}$ ), or even more, may be needed to make the color of the unknown of approximately the same intensity as the standard. The colorimeter readings should be made without delay, the standard prism being set at the 10 or 15 mm. mark.

---

\* The solution may be prepared by dissolving 0.944 Gm. (14.5 grs.) ammonium sulphate or 0.764 Gm. (11.7 grs.) ammonium chlorid of the highest purity in distilled water, and making up to 1000 mils (33.3 f $\bar{3}$ ).

† For the modified Nessler's solution place 100 Gms. (3.2 f $\bar{3}$ ) mercuric iodid and 50 Gms. (1.6 f $\bar{3}$ ) potassium iodid, both finely powdered, in a liter (quart) volumetric flask and add about 400 mils (13.5 f $\bar{3}$ ) water. Now dissolve 200 Gms. (6.4 f $\bar{3}$ ) potassium hydroxid in about 500 mils (16.6 f $\bar{3}$ ) water, cool thoroughly and add with constant shaking to the mixture in the flask; then make up with water to the liter mark. This usually becomes perfectly clear. Keep at body temperature over night, or until the yellowish white precipitate, which may settle out, is thoroughly dissolved, and only a small amount of dark, brownish-red precipitate remains. The solution is now ready to be siphoned off and used.

**Creatinin.** About 10 mils (2.7 f3) of blood are drawn directly from a vein into a small bottle containing a little powdered potassium oxalate or 5 drops (0.3 mil) of a 20 per cent. solution to prevent clotting. Six mils (1.6 f3) of the well-mixed blood are treated with 24 mils (6.4 f3) of water (4 vol.). After the corpuscles have been laked, about 1 Gm. (15.4 grs.) of dry picric acid is added and the mixture stirred at intervals with a glass rod until it is light yellow. When the protein precipitation is complete, the mixture is centrifuged and the supernatant fluid filtered through a small 7 cm. (2.7 in.) filter paper. From 17 to 21 mils (4.5 to 5.6 f3) of filtrate are usually obtained. To 10 mils (2.7 f3) of the filtrate is added 0.5 mil (8 m.) of 10 per cent. sodium hydroxid, and a similar amount of alkali added to 10 mils (2.7 f3) of standard creatinin in saturated picric acid (containing 0.2, 0.5 or 1.0 mg. (0.003, 0.007 or 0.01 gr.) creatinin to 100 mils (3.3 f3) of picric acid). A standard solution of this creatinin, 1 mg. to 1 mil (0.01 gr. to 16 m.) is kept in 0.1 N hydrochloric acid. From this may be prepared a stock solution of picric acid, 5 mgs. to 100 mils (0.07 gr. to 3.3 f3) by diluting 5 mils to 100 mils (1.3 to 3.3 f3) with saturated picric acid. By pipetting 0.4, 1.0 and 2.0 mils (7, 16.2 and 32.4 m.) of this solution into 10 mils (2.7 f3) graduates with a Mohr pipette, and diluting to the mark, standards of the above strength are prepared. For the Duboscq colorimeter the standard prism can be set conveniently at the 15 mm. mark.

The estimation of the creatinin may likewise be carried out with the use of the Autenrieth-Königsberger colorimeter of Hellige. In this case less blood is necessary. Two mils (32.4 m.) are treated in a cylindrical centrifuge tube with 8 mils (2.1 f3) of water and other manipulations as above. For the determination proper, 0.1 mil (2 m.) of 10 per cent. sodium hydroxid are added to 2 mils (32.4 m.) of the picric acid filtrate in a small test-tube. Simultaneously 1 mil (16 m.) of the alkali is added to 20 mils (5.3 f3) of a saturated solution of picric acid containing 1.5 mgs. (0.02 grs.) creatinin to 100 mils (3.3 f3) to serve as standard for the wedge. At the end of ten minutes the wedge is filled with the standard, the cup with the unknown, and readings made. The following formula, in which R represents the colorimetric reading and 5



the dilution, will give the results expressed in milligrams per 100 mils (3.3 f $\bar{3}$ ) blood.

89 —  $R \times 0.0179 \times 5 = \text{mg. creatinin in 100 mils (3.3 f}\bar{3}\text{) blood.}$

Creatinin values from 2.5 to 3.0 mgs. (0.03 to 0.04 grs.) per 100 mils (3.3 f $\bar{3}$ ) of blood should be viewed with suspicion, and figures from 3.0 to 5 mgs. (0.04 to 0.07 grs.) should be regarded as decidedly unfavorable, while an amount over 5 mgs. (0.07 grs.) probably indicates an early fatal termination.

A search for the etiologic factor or factors in each case of chronic interstitial nephritis is of great importance, because the removal of such a cause is essential, if one hopes to check the course of the disease. A study of the patient's occupation, habits, food, etc., must be made in order to discover and remove anything that is capable of increasing the normal work of the kidneys, or that may act as a renal irritant. Disorders of digestion and metabolism, sometimes indicated by indicanuria and oxaluria, must be eliminated. All possible foci of infection, such as the teeth, gums, sinuses, tonsils, prostate, bladder, and so forth, must be investigated. Those whose occupation exposes them to the danger of intoxication with lead, mercury, and poisonous fumes must change their work. Any fault which interferes with proper digestion must be removed. The teeth must be put in the best condition possible to insure proper mastication.

Physical overexertion must be avoided in order to spare the heart, which is practically always hypertrophied in chronic interstitial nephritis, and also to relieve the kidneys of the necessity of eliminating the resultant surplus of waste products. Mental overwork is as injurious as physical overexertion. Absolute rest in bed is not only unnecessary, but injurious if long continued, except in the terminal stages of the disease. The patient should be permitted to be about doing a fraction of his normal amount of work. Loss of sleep and fatigue, due to social activities, must be avoided. In short, the patient must conserve strength and energy, and, speaking generally, his activities must be reduced by one-fourth or one-half. Such games as tennis, baseball and swimming must be forbidden. Moderate walking and golf in moderation are



beneficial. Nine to ten hours in bed each night, and, in addition, in certain cases, a siesta should be advised.

Patients with chronic interstitial nephritis do best in a warm, dry climate, because in such a climate the activity of the skin lessens the work required of the kidneys. In ordering a patient to such climate, however, one must take under consideration a number of factors, such as the quality of the food to be obtained, the altitude, the character of the diversions offered, the season of the year, and similar details. It is also to be remembered that the patient seldom goes alone to the place selected, but takes one or more members of his family. If one who is unable to find his own diversion and cannot interest himself is sent to a place where there are few people and less of the things that attract and interest him, the mental unrest and unhappiness would nullify any advantage expected to be derived from climate. Likewise, if the creature comforts are poor, the resultant discomfort may prevent the obtaining of sufficient benefit to justify the expenditure of the time and money.

A climate that is suitable throughout most of the year is found in southern California and Hawaii. The winter climate of southern Texas, Arizona, and New Mexico, in this country; Naples, Algeria, and the Island of Capri abroad, are suitable. As practically every case of chronic interstitial nephritis is associated with cardiac hypertrophy and hypertension, much consideration must be given to the altitude of the resort to be selected. Many patients who are very comfortable at elevations up to 800 or 1000 feet become dyspneic on the slightest exertion and suffer from cardiac palpitation at higher altitudes. Permitting a patient with considerable myocardial change to go to a high altitude may result in death. It is plain to be seen that, because of the many factors which must be considered, no one place can be selected to suit all patients, and the choice is made largely to suit the individual requirements. A patient may be very much better off at home where there are advantages of good food and comfortable surroundings, even though the climate is severe, than in a warm, dry climate, with poor food, away from friends, and suffering the tortures of homesickness.

Except during an acute exacerbation, or when uremia

occurs, an exclusive milk diet should not be prescribed, for the reason that the food value is too small as compared with the bulk of liquid. The nephritic patient who is up and around requires about 2000 calories. To supply this would require 5500 mls ( $5\frac{1}{2}$  qts.) of skimmed milk or buttermilk, or about 2750 mls ( $2\frac{3}{4}$  qts.) of whole milk. The transporting of this quantity of fluid by the circulation throws additional work on a vascular system that is already severely taxed. It is better to supply the required calories by a mixed diet. The proteins must be reduced, usually to about 80 Gms. ( $2\frac{1}{2}$  ozs.). As it does not matter in what form of food the protein is supplied, there can be no objection to the taking of a small quantity of meat. The quantity of protein-bearing food is the important thing, and this quantity may be roughly estimated by remembering the following values: The average helping, 3 tablespoonsful (45 mls), of peas and beans contains from 3 to 6 Gms. (46.3 to 92.6 grs.) of proteins; other vegetables up to 3 Gms. (46.3 grs.). A slice of bread  $3\frac{1}{2}$  inches (8.8 cm.) square and  $\frac{1}{2}$  (1.2 cm.) thick contains 3 to 4 Gms. (46.3 to 61.7 grs.) of protein. An average helping (one slice) of the meats contains 20 to 30 Gms. (308.6 to 462.9 grs.). Three ounces (93.3 Gms.) of fish, which is an average helping, contain 10 to 20 Gms. (154.3 to 308.6 grs.); one egg contains 6 to 7 Gms. (92.6 to 108 grs.). The protein intake must be varied according to the nitrogen content of the blood, as shown by the blood tests for uric acid and urea. An increase in their retention indicates a diminished renal efficiency, and demands decrease in the protein intake.

Simple, easily digested food should be eaten in moderate amounts and with regularity. It should be well masticated. Rich and highly seasoned foods should be avoided. Common-sense-rules concerning the restriction or avoidance of cakes, pies, rich puddings, and candies must be obeyed. Meat occasionally should be permitted, fish, oysters and chicken being preferable to the red meats. The feces should be examined, and if it is shown by the fermentation test that the proteins or carbohydrates are not being properly digested, the diet must be arranged accordingly. Frequent urinalyses should be made for the detection of indican and scatol, which indicate absorption of putrefactive materials from the colon.

When present, the question of diet, the manner of mastication, and the ability of the digestive juices to handle the food must be investigated.

The intake of fluid should be restricted in a measure. There is not the same need for washing out the tubules in chronic interstitial nephritis that there is in the chronic parenchymatous type, and, besides, the heart and vessels must not be overworked by the necessity of carrying large amounts of fluid. In the average case the quantity of fluid should be restricted to 3 pints (1420 mls) a day.

Alcohol, particularly gin, is to be forbidden; or, at least, very greatly restricted. In some instances a small amount of wine may be permitted with meals. Tobacco, because of its poisonous action upon heart and vessels, should be greatly restricted, and, when possible, quite proscribed.

The skin must be kept in good condition, because of its importance as an organ of elimination. A warm cleansing bath, using a bland soap, such as pure castile, should be taken each night before retiring. Great care must be taken to avoid chilling during or after the bath. Cold baths are contraindicated. In the early stages of the disease, during warm weather, a cool sponge bath will be refreshing, and will do no harm. The skin must always be protected against sudden changes of temperature, and in cool and cold weather woolen underwear should be worn.

Throughout most of the course of chronic interstitial nephritis the heart is gradually hypertrophying to carry the gradually increasing burden thrown upon it. During this process no direct cardiac treatment is indicated. The patient's mode of life must be regulated so as to avoid all unnecessary circulatory strain. In many cases a time comes when the heart can no longer compensate, and dyspnea, edema, cyanosis, and other signs of decompensation become evident. Rest, both physical and mental, is then imperative. Unless dyspnea prevents, the patient should be kept in bed, while the amount of ingested liquids is reduced and cardiac stimulation is employed. Digitalis, in doses sufficient to produce a perceptible impression on the heart, should be given. The tincture in doses of 5 to 20 drops (0.31 to 1.2 mil) every four hours or an infusion freshly made from assayed leaves 2 to 4 drams

(7.5 to 15 mils) every three or four hours are the best forms in which to administer the drug. Its action must be closely observed, and if the heart becomes arrhythmic or irregular, the use of the drug must be abandoned. An efficient tincture of *strophanthus* is often of great value. The dose for each individual must be found by trial, as there is a wide difference in patients as to their ability to take the drug. In some, small doses taken for a few days produce nausea, vomiting and diarrhea. The initial dose should be small, 3 drops (0.15 mil) every four hours, increased as rapidly as possible, but seldom exceeding 10 drops (0.62 mil). The administration of tincture *nux vomica* in 15- to 25- drop (0.93 to 1.56 mils) doses often yields very excellent results. To tide a patient over a cardiac crisis, strychnin nitrate,  $\frac{1}{30}$  to  $\frac{1}{20}$  grain (0.002 to 0.003 Gm.), or camphor, 1 to 3 grains (0.06 to 0.19 Gm.), in 10 to 20 drops (0.62 to 1.2 mils) of sterile olive oil, may be given hypodermically. Venesection is at times of great value in extreme hypertension or when the heart is dilated. Ten or more ounces (300 or more mils) of blood should be abstracted, save in the face of a severe anemia. The results secured must determine the question of its repetition.

The systolic blood-pressure is almost always considerably higher than in health. Rarely it may be as high as 320 mm. Unless the systolic pressure be excessive, *i.e.*, over 200 mm., it requires no treatment, as a hypertension of this sort is largely compensatory. In many of these patients unpleasant symptoms are experienced when the blood-pressure is reduced to 150 mm. or lower. When the pressure is excessive there is danger of retinal hemorrhage, cerebral hemorrhage, and acute dilatation of the heart, and it, therefore, requires treatment.

The use of drugs to lower the pressure is very unsatisfactory, for their action is evanescent. Nitroglycerin is very popular in the treatment of hypertension, but in the doses given ordinarily it is incapable of doing any good. When the dose is sufficiently large to make an impression on the vascular hypertension it produces headache and dizziness. Sometimes by the use of this drug headache is produced without any observable effect on the hypertension. Sodium nitrite, 1 to 5 grains (0.06 to 0.32 Gms.), is sometimes given.



Like nitroglycerin, its action is uncertain, and in most cases it does no good, and often causes indigestion. The same is true of sodium or potassium iodid. In regard to the iodids, it is to be remembered that some patients have a marked idiosyncrasy toward them. Edema of the fauces and uvula sometimes occurs, even with very small doses. In one patient one drop of the saturated solution of sodium iodid thrice daily resulted in edema of the uvula. The iodine is excreted by the kidneys; therefore, the possibility of harming these organs must be kept constantly in mind. The iodid of sodium is to be preferred to the iodid of potassium.

The most efficient and safest means of reducing the pressure is by elimination through the skin. For this purpose sweat-baths are used, as described in discussing the treatment of acute nephritis (see page 586). The use of diaphoretic drugs is to be avoided, except in those rare cases where sweating cannot be induced by the various hot packs and baths.

The bowels should be made to move two or three times daily for a day or two by the use of the phosphate or sulphate of sodium, and then once daily for several days. Brisk purging is seldom to be resorted to in the treatment of hypertension.

Dyspnea, especially of the nocturnal type, is often very distressing, especially when associated with mental excitability and restlessness. Opium,  $\frac{1}{2}$  to 1 grain (0.03 to 0.06 Gm.) in a suppository, or morphin,  $\frac{1}{8}$  to  $\frac{1}{4}$  grain (0.008 to 0.01 Gm.) hypodermically, is the most efficient treatment, but is contraindicated in uremia. When the dyspnea and restlessness are not very marked, sodium or potassium bromid, 10 to 30 grains (0.6 to 1.9 Gm.), or chloral, 5 to 10 grains (0.32 to 0.6 Gm.), may be used, but are very likely to cause nausea, and, perhaps, vomiting.

Cerebral hemorrhage, even though slight, demands immediate and absolute rest in bed. Absolute rest of body and mind is very essential, and must be secured with opium or morphin, if necessary. If the patient is seen at the beginning of the hemorrhage, venesection should be done immediately, and as much blood as safety permits should be withdrawn. If hypertension exists, venesection is of value, even some hours after the occurrence of the cerebral hemorrhage. Vis-



itors should be excluded from the room, and all noises reduced to a minimum. Purging should be avoided for the first forty-eight hours, because of the importance of rest as an aid to the cessation of bleeding. Later the bowels should be kept open. The food should be small in quantity and concentrated. The intake of liquids must be greatly reduced. Stimulants must be avoided. After sufficient time, usually several days, has elapsed to insure the formation of a firm clot in the ruptured vessel, the amount of liquid consumed may be very gradually increased. Subsequently, general massage is of value. When the ability to move the part is regained, daily exercise for the parietic muscles should be advised with faradism.

When uremia develops, food should be withheld, and later the diet should be limited to milk or skim milk, of which 6 ounces (180 mls) may be given every four hours. The patient must remain in bed, and active elimination through the skin and bowels begun. Sweating should be induced by means of hot, wet, or dry packs, hot-air baths, or the electric light bath, as described on page 586. These should be given once, and in urgent cases, even twice each day. Five or six watery bowel movements should be secured by the use of phosphate or sulphate of sodium. Venesection may be very helpful. Recently one of the authors saw a young adult woman with acute parenchymatous nephritis following pregnancy, who suddenly became uremic with white, profusely sweating skin, moderately dilated pupils, dark, cyanotic lips and nails, rapid pulse, and sixty respirations per minute, with pulmonary congestion and edema. Death seemed imminent. The prompt removal of 27 ounces (810 mls) of blood was followed by immediate amelioration of symptoms and later by recovery. From 10 to 30 ounces (300 to 900 mls) of blood should be withdrawn. If the kidneys are capable of eliminating water this should be freely supplied by mouth, if the patient is conscious and the stomach is retentive, or per rectum, by means of the Murphy drip or continuous enteroclysis, as described on page 583. Lumbar puncture is sometimes of benefit (*v. s.*).

### AMYLOID DEGENERATION OF THE KIDNEYS.

The exact nature of this affection is still unknown. Rokitsansky, in 1842, first described the rather characteristic appearance of the disease in the kidneys and thought the condition one of lardaceous or fatty infiltration. Virchow considered the substance as related to amyloid and cellulose, and gave it the name of amyloid, which is still in use, although it is definitely known that the substance is not amyloid material. The manner in which the change in the kidneys is brought about is not known.

It seems to follow in the wake of infections, and is considered a secondary disease. The degeneration is usually found also in the liver and the spleen, and is most commonly encountered in pulmonary tuberculosis, especially when characterized by cavity formation and an abundant purulent expectoration. It is sometimes seen in cases of bronchiectasis.

In protracted suppuration in any part of the body, but especially of bones and joints, it is often seen. Syphilis, either acquired or hereditary, is a common cause. It may occur in association with chronic parenchymatous or chronic interstitial nephritis.

The kidneys are usually larger than normal, unless the degeneration occurs in kidneys that have been contracted by chronic interstitial nephritis. They are pale with opaque, yellowish-white mottlings, and the capsule strips easily, leaving a smooth surface. On section the tissue is gray and translucent, except in the yellow, opaque areas, where fat globules and dead cells are accumulated. The glomeruli are visible and prominent as translucent points. The amyloid deposit acquires a reddish brown or mahogany color on the application of Lugol's iodine solution. Microscopically, the deposit is found chiefly about the blood-vessels and in the glomeruli. The epithelial cells of the tubules show advanced degeneration.

In many instances the symptoms produced by the primary disease, *i.e.*, tuberculosis, syphilis, suppurative process, completely overshadow the symptoms produced by the degeneration in the kidneys. As a rule, the urine is abundant in quantity, clear, pale yellow in color, acid, and has a low specific

gravity. The albumin percentage is high, and there is very little sediment, which contains only a few hyalin, fatty and sometimes waxy casts, an occasional leucocyte, and very few epithelial cells.

Dropsy is a frequent accompaniment of amyloid disease. Probably the greatest factor in the production of edema is the cachexia of the primary disease. Diarrhea is not infrequent, and may be due to renal insufficiency (uremia), or to amyloid disease of the intestinal mucous membrane.

The therapeutic measures to be adopted relate to the active treatment of the primary disease, rather than to the amyloid lesion *per se*. In suppurative conditions, such as empyema or abscesses in any part of the body, the pus must be given a free outlet, and every method known to modern surgery employed in securing prompt healing. If the primary disease is tuberculosis or syphilis, active treatment of this may prevent the occurrence of amyloid degeneration.

General hygienic measures, such as the care of the skin, the securing of fresh air and plenty of sleep, are, of course, essential. Food should be given in accordance with the digestive powers of the patient. The object should be to secure the maximum nourishment possible.

## NEPHROLITHIASIS.

By this term is meant the precipitation in the kidney substance or in the renal pelvis of urinary solids, forming concretions that vary in size from sand grains to large concretions or calculi. Most commonly they are formed of uric acid, with some sodium and ammonium urate and a small quantity of xanthin. The uric acid stones are usually yellowish, reddish brown or brick-red in color, rather smooth, most often rounded and hard. The uric acid nature of the calculus may be discovered by treating some of the powdered stone with nitric acid in a porcelain dish and evaporating to dryness. After cooling allow a drop or two of ammonia water to come in contact with the residue. Uric acid or urates will give a light blue or violet color.

Next in the order of frequency are the stones composed of calcium oxalate. They are generally of a dark gray, almost

blackish color. They are extremely hard and may occur as small, smooth concretions, or as medium-sized or large stones with a very rough surface, generally called a mulberry calculus. The roughened surface is due to the protrusion of the sharp-pointed octahedral crystals of calcium oxalate, which crystals lacerate the tissue and produce early and severe symptoms. When some of the powdered stone is gently heated, then treated with hydrochloric acid, it effervesces.

Phosphatic calculi, which consist in the main of calcium phosphate and ammonium-magnesium phosphate, are uncommon in the kidneys. The formation presupposes ammoniacal decomposition of the urine, which is rare in the kidneys, but common in the bladder, and is usually due to infection with various micro-organisms. These stones, when found in the kidney, are usually in association with the presence of pus. If pyogenic infection of the kidneys takes place after a uric acid or an oxalate stone has formed therein, these calculi will be coated with a layer of phosphates. The surface of phosphatic calculi is generally rough, but occasionally it may be rather smooth. They may be white or gray or tinted yellow. The stone, when powdered, dissolves in acetic or hydrochloric acid.

The cause of the formation of renal calculus is not clear. That the appearance of the urates, uric acid, oxalates, etc., in the kidneys in large amount is due to disorders of metabolism is undoubted. Osler states that sedentary occupations seem to predispose to stone. The influence of diet is not definitely known, but it is probable that when purin bodies in large quantity are ingested, and because of insufficient exercise or other cause are improperly oxidized, uric acid or urate stones may develop. The same is true of the ingestion of foods rich in oxalic acid. Nephrolithiasis may occur at any age, but it is most commonly encountered in the fourth decade of life. Holt states that uric acid deposits in the kidneys of children are very common. Males are more frequently affected than females.

The kidney may remain quite healthy, even in the presence of a calculus, but generally a diffuse nephritis is present. If the ureter is obstructed, hydronephrosis develops; and, if an infection occurs, a pyonephrosis results, in which event

the renal destruction may be so great that the kidney is converted into a large, irregular, fluctuating mass with thick walls. The number of stones varies from one to many. They are usually found in the pelvis or calices; less commonly they are imbedded in the substance of the kidney. It is not uncommon for renal lithiasis to be bilateral.

A stone or stones may remain in the kidney for years without producing any symptoms until the existence of a pyogenic infection is betrayed by chills, fever and sweats with pyuria. In most instances, however, a dull aching pain, sometimes scarcely more than a discomfort, in the lumbar region is noted. At times the pain becomes colicky in character and radiates downward and forward to the groin, and often into the testicle. Sometimes it is referred to the head of the penis. The pain may occur very suddenly, and be so extremely severe that beads of sweat appear on the forehead. During the paroxysm the patient may be unable to move, but usually he tosses wildly about, and rolls on the floor shrieking and groaning. Pain of this character is spoken of as renal colic, and is often accompanied by nausea and vomiting. The intense pain lasts for several hours, as a rule, unless treatment is instituted, and passes away rather abruptly, sometimes suddenly. It is often followed by soreness, and at times by prostration. After the attack the first urine voided may contain macroscopic blood. Nearly always microscopic blood will be found. Occasionally anuria occurs and may become a very serious symptom. The mortality rate of calculus anuria is very high.

W. F. Braasch and A. B. Moore<sup>11</sup> state that of the 742 cases of lithiasis of renal origin operated on at the Mayo Clinic up to June, 1915, 512 stones were removed from the kidney, and 230 were found lodged in some portion of the ureter. In ureteral stone, the pain, which is due either to intrarenal tension as a result of urinary obstruction, or because of localized infectious changes, was referred largely to the renal region in 67 per cent. of the cases; to the upper abdominal quadrant in 15 per cent.; to the region of the lower ureter in 9 per cent.

Renal colic is not diagnostic of stone, as it may occur in renal hemorrhage due to the passage of clots, or as a result



of obstruction of the ureter from other causes. An *x*-ray examination is of great aid in the diagnosis.

Calculi containing calcium throw a shadow, but 50 per cent. of stones are not revealed by Röntgen-rays for various reasons, such as obesity, embedding of a stone in fat, soft structure of a stone, or its obstruction by the iliac artery. Conversely, all shadows shown in the region of the kidney by the *x*-rays are not due to stones, but may have been caused by glands, calcareous deposits, or phleboliths.

In all cases where the *x*-ray evidence is doubtful the ureteral catheter must be used. W. F. Braasch<sup>12</sup> states that 80 per cent. of ureteral stones were detected by the catheter, and 70 per cent. of stones in renal pelvis were thus detected.

The frequent or continuous presence of urates, uric acid and oxalates in the urinary sediment between the attacks of colic, or associated with dull aching in the lumbar region, is rather good circumstantial evidence in favor of the diagnosis of renal calculus. Many years ago one of the authors saw a man of gouty diathesis, aged 60 years or more, who presented no definite symptoms of renal disease excepting polyuria leading to the suspicion of contracted kidney. He died suddenly of uremia. Each kidney was transformed into a large bag containing enormous stones which, weighing about half a pound (0.2 Kg.), occupied the pelvis and calices. The substance of the kidney was reduced to a mere rim  $\frac{1}{4}$  of an inch (6.3 mm.) in thickness. These stones had existed for years, and produced absolutely no symptoms.

As a rule the physician is first called in to treat the renal colic. If the attack is mild a hot bath will relax the spasm and end the attack. The patient should be placed in a tub of water of sufficient depth to cover the legs and trunk. The temperature of the water should be 100° F. (37.7° C.), which, after the patient has been in the tub a minute or two, should be raised, in the course of ten to fifteen minutes, to 110° or 115° F. (43.3° or 46.1° C.), depending upon the ability of the patient to endure heat. While in the bath an ice-bag or cloth wrung out of very cold water should be placed on the head. The bath should be discontinued if faintness occurs. After the bath the patient is quickly but gently dried and placed in bed between blankets. It is important that during this treatment

the patient be as passive as possible. He should not dry himself, and all his movements should be slow and gentle. A hot-water bottle should be applied to the site of pain after the patient is in bed, or, still better, hot fomentations should be used. These consist in the application of towels wrung out of boiling water, and applied to the site of pain. Care must be taken to wring them quite dry to avoid scalding the skin. The towels must be removed as often as necessary (usually every three to five minutes) to maintain the heat. Poultices are distinctly inferior, as they are not only slowly made, but quickly lose their heat, after which they add to the discomfort of the patient. The materials that enter into the composition of the poultices are valueless in the treatment of colic, the sole value of the poultice residing in the heat.

If the pain continues to increase, or if the attack is severe from the onset, a hypodermic injection of morphin,  $\frac{1}{4}$  grain (0.01 Gm.), and atropin,  $\frac{1}{100}$  grain (0.0006 Gm.), must be administered. It is best to begin with  $\frac{1}{4}$  of a grain (0.01 Gm.) and repeat in fifteen or twenty minutes if ineffectual. These patients tolerate large doses of morphin, and the drug must, therefore, be used for effect, without regard to dose. Atropin, because of its ability to inhibit contraction of unstriated muscle, is of distinct value. It should be given in doses of  $\frac{1}{150}$  of a grain (0.0004 Gm.), and repeated as often as necessary, watching closely, however, to avoid toxic effects.

The use of chloroform to quiet the patient is not advisable, because of the possibility of the danger of disturbing the action of an already functionally deranged kidney. The likelihood of its favoring the occurrence of anuria, so fatal in renal lithiasis, also is to be recalled.

If collapse occurs, aromatic spirits of ammonia should be given by mouth or by inhalation, and, if necessary, camphor, 5 grains (0.3 Gm.) in oil, or strophanthin (Merck's),  $\frac{1}{120}$  to  $\frac{1}{60}$  grain (0.0005 to 0.0011 Gm.) should be given intravenously.

Should no urine be voided within a few hours of the attack, examination should be made to determine whether this is due to retention or suppression. If due to suppression, draughts of hot lemonade should be given and more heat applied to the lumbar regions. Anuria lasting more than

twenty-four hours is serious, and surgical intervention becomes necessary.

The mortality rate is much lower in those cases that are promptly operated upon. Continuous enteroclysis of normal salt solution or 2 per cent. dextrose solution is indicated. Ten grains (0.6 Gm.) of theobromin-sodium-salicylate (diuretin) every four hours is often of value.

After the attack of renal colic, the case must be carefully studied, with the object of determining the functional capacity of each kidney by the phenolsulphonephthalein test. This necessitates differential catheterization, and no operative treatment must be considered until after this information is at hand. The urine must be repeatedly examined, and if uric acid, urates, or oxalates are found, the diet must be arranged so as to diminish the intake of the bodies that form these compounds. Water must be administered to flush the kidneys freely. To prevent, so far as possible, the occurrence of infection, hexamethylene-tetramin (urotropin), 5 to 10 grains (0.32 to 0.65 Gm.), two or three times daily should be given. It must be remembered that hematuria may follow the too prolonged administration or the giving of too large doses of this drug.

If the patient has had but one attack of colic and can be kept under close observation so as to be assured that infection is not taking place, operation may be indefinitely postponed and often avoided. If pus begins to appear in the urine and differential catheterization shows that it comes from a single kidney, operation is indicated, provided the phenolsulphonephthalein test shows these organs to be functioning sufficiently. If a calculus be passed, symptoms disappear, and an *x*-ray examination shows no shadows, the operation should be postponed until symptoms arise to indicate definitely the presence of another stone. If an *x*-ray examination reveals several shadows in the kidney or ureter, operation is advisable.

Before an abdominal operation is attempted, the passage of the stone in the ureter may be aided by (1) catheter manipulation; (2) injection of sterile glycerin or oil; (3) fulguration; (4) ureteral dilatation; (5) cutting of the meatus, and (6) the use of ureteral forceps.

It has been claimed that the injection of oil and glycerin into the ureter would both increase the peristalsis and lubricate the walls of the ureter so that the stone would slip out, but this theory, however, is not corroborated by clinical observation. It is difficult to conceive how the natural efforts of peristalsis or how the natural lubrication in the ureteral mucosa could be improved on.<sup>13</sup>

### HYDRONEPHROSIS.

By hydronephrosis is meant the overdistension of the renal pelvis with urine. The disease is usually confined to one kidney, and is due to obstruction somewhere along the course of the ureter. Bilateral hydronephrosis is very rare. It may occur as the result of a new growth compressing both ureters or occluding both ureteral orifices in the bladder. Hydronephrosis may be either congenital or acquired. The etiological conditions responsible for the acquired type are (1) stricture following inflammatory and traumatic lesions; (2) blockage of a ureter by a calculus; (3) compression of the ureter by a tumor; (4) constriction of the ureter by fibrous bands, due to an inflammatory process in neighboring tissues. W. F. Braasch<sup>14</sup> states that the etiological conditions commonly observed in congenital cases are (1) anomalous renal blood-vessels; (2) atresia of the ureter near the renopelvic juncture, and (3) displaced kidneys. Of 27 cases studied at the Mayo Clinic, 20 were associated with an anomalous blood-vessel. Usually such a blood-vessel is given off from the aorta or renal artery, and, passing the ureter anteriorly, enters the kidney near the lower pole.

The presence alone of an anomalous vessel is insufficient to cause hydronephrosis, as the ureter and the anomalous vessel are not usually in contact. It is probable that the loss of perirenal fat permits enough sagging of the kidney to cause the ureter to bend over the vessel to form a kink. The chief symptom is pain in the upper lateral aspect of the abdomen. The pain is not referred, as in renal or ureteral calculus, but remains localized, and though severe, it is less so than the pain due to renal stone. It is usually acute for hours, and followed by a soreness that may persist for sev-

eral days. Quite commonly nausea and vomiting accompany the pain. If the hydronephrosis be sufficiently large, a tumor may be palpable, which may suddenly disappear, due to the escape of urine through the ureter. A radiogram made after the injection of argyrol and bismuth solutions into the renal pelvis is of great aid in the diagnosis, and is especially valuable in distinguishing between lithiasis and nephrosis. A history of polyuria following the attack of pain is always suggestive.

Cystoscopic examination and catheterization of the ureters is of great assistance in the diagnosis. The capacity of the renal pelvis may be measured by injecting boric acid solution stained with methylene blue, the dye being used to determine whether there is any return flow alongside the catheter. The normal capacity of the renal pelvis is 5 to 15 mls (1.3 to 4 f5). Sometimes as much as 30 mls (1 oz.) may be injected before pain is caused. If 40 mls (1.3 oz.) or more can be tolerated without pain, a diagnosis of dilatation of the pelvis is justified.

When pain is severe, morphin,  $\frac{1}{8}$  to  $\frac{1}{4}$  grain (0.008 to 0.01 Gm.), may be necessary, and hot applications are often soothing. As a rule, it is necessary to relieve the obstruction of the ureter by surgical measures. In those cases due to kinking of the ureter, the kidney should be anchored in its normal position. When the renal substance is almost entirely destroyed, and when infection has taken place, the entire kidney should be removed.

### PYOGENIC INFECTIONS OF THE KIDNEYS.

Under this heading are considered pyelitis, pyelonephritis, suppurative pyelonephritis, suppurative nephritis, and pyonephritis.

By pyelitis is meant an inflammation of the renal pelvis; if the substance of the kidney is also involved, the term pyelonephritis is used. If miliary abscesses are scattered throughout the kidney substance and in the pelvis, the condition is spoken of as suppurative pyelonephritis; and if the abscesses are limited to the renal substance, it is termed suppurative nephritis. If the suppurative inflammation results



in the formation of one or more fairly large circumscribed collections of pus in the renal substance, it is called abscess of the kidney. When, from ureteral obstruction to the escape of urine from the pelvis of the kidney, distension occurs with subsequent infection, the change in the organ is called pyonephrosis.

Many varieties of micro-organism are capable of producing these pyogenic inflammations, but the bacteria most commonly found are the colon bacillus, the staphylococcus, and the streptococcus. The incidence of the colon bacillus is more frequent than that of the other two micro-organisms. The bacteria may reach the kidneys through the blood—hematogenous infection—from a focus elsewhere in the body, as infected tonsils, abscesses about the teeth or elsewhere, and as a result of pyemia, septicemia and ulcerative endocarditis. As a result of fecal stasis in the colon, the colon bacilli may find their way into the intestinal wall and be conveyed to the kidneys by the blood. The infection may be urogenic, *i.e.*, arising from an ascending infection from the bladder or ureter. This is observed in cystitis, especially in individuals with low resistance and relaxed musculature. Obstruction of the ureter by a twist or by a calculus, or as the result of compression by a neoplasm, followed by infection converts a hydronephrosis into a pyonephrosis. Pyogenic infection of organs or tissues in the vicinity may extend to the kidney, and, rarely, renal infection is the result of a penetrating wound.

When the renal pelvis alone is affected the pathologic picture is variable, depending upon the length of time the infection has persisted, and the virulence of the infecting organism. As a rule, staphylococcus infections are relatively mild, while infections with the streptococcus may be very severe. In acute pyelitis there is marked congestion and swelling of the mucous membrane, with pus cells, and, sometimes, red-blood cells upon its surface.

In some of the severe types of streptococcus infections, punctiform hemorrhages are observed. In chronic inflammation the mucous membrane is much thickened, brownish or grayish in color, and ulcerations are commonly present. When the infection occurs after obstruction of a ureter, the

wall of the pelvis may be thinned from overdistension, and a deposit of urinary crystals, usually phosphates, takes place. The contained urine is usually turbid and foul-smelling.

Extension to the renal substance is indicated by grayish lines radiating upward from the papillæ, which lines, under the microscope, are found to be composed of pus cells and bacteria. The microscope may likewise reveal areas of round cell infiltration. Miliary abscesses may be seen scattered through the cortex and medulla. In some instances, by fusion of the miliary abscesses, larger ones are formed. Unless the infection be hematogenous, usually but one kidney is infected, the unaffected organ becoming hypertrophied in chronic cases, to compensate for the gradually disappearing function of its diseased mate. One of the authors has observed in a patient, dead of typhoid fever, an impacted stone in the left ureter near the bladder wall, causing complete obstruction, which had evidently existed for years. The kidney was shrunken and the pelvis and calices slightly dilated. The right kidney was about twice the normal in size and structure, and no symptoms of renal inadequacy or disease were present.

Pyogenic infections of the kidney may exist with no symptoms, or the symptoms may be slight and completely overshadowed by those of the primary infection, such as cystitis or septic endocarditis. The principal subjective signs are pain, swelling, and constitutional disturbances with urinary abnormalities. The changes in the urine are most important. Pus cells are practically always found, but may be so few as to be detected only by microscopic examination of the urine, or may be sufficient in number to be seen by the naked eye as pus. Sometimes they are absent. In severe acute and chronic cases blood may appear. Bacteria are often found in abundance. Albumin is present, usually in amounts larger than can be accounted for by the presence of pus. Casts may be present, but unless a nephritis coexists they are likely to be few or absent. Leucocytic or pus casts may be found. The reaction of the urine depends upon the invading type of micro-organism. In colon bacillus infections the urine is usually acid. Staphylococcus infections are likely to produce an alkaline urine, in which event the odor will be

ammoniacal, and the sediment will contain a large number of amorphous and triple phosphates.

Pain, when present, is usually not severe, and is complained of by the patient as a dull ache in the lumbar region. Deep pressure over the affected kidney may reveal tenderness. Sometimes acute exacerbations of pain occur, and attacks of colic may be produced by the passage of clots of blood, or of plugs of tough mucus.

In pyonephrosis a tumor mass may be felt, which is usually tender on pressure. In most other pyogenic infections of the kidney the organ is not palpable.

The classic constitutional symptoms of the presence of pus (chills, fever and sweats) may be present. Sometimes only occasional slight chilliness is noted at irregular intervals, and the temperature may never exceed 99° F. (37.2° C.). Sweats may occur without the association of chills and fever. On the other hand, intermittent fever with a range of 70° F. (21.1° C.) may occur, with rigor and sweats. As a rule there is a loss of appetite and a feeling of malaise. Usually a positive diagnosis cannot be made without a cystoscopic examination and ureteral catheterization.

If the etiology of pyogenic infections of the kidney is borne in mind, much may be accomplished in prophylaxis by removal of the original focus of infection. Therefore, infected tonsils, abscesses about the roots of teeth, chronic sinus infections, and other potential causal factors of sepsis must be removed; and important in this connection is the fact that collections of pus deep in the body of the tonsil or abscesses at the apices of the teeth usually do not drain towards the surface, and therefore the absorption of the infectious organism is enhanced. It is also to be remembered that commonly these original foci of infection give rise to no symptoms, or to such minor symptoms as to escape the patient's attention. The absence of symptoms does not guarantee that such a focus will not produce far-reaching evil effects in the body.

Fecal stasis, often causing chronic or recurrent headaches and other symptoms of intestinal toxemia, favors the infection with the colon bacillus, and, therefore, a torpid bowel is to be energetically treated, not only with the view to relieving the immediate expression of the toxemia, but also to prevent

the occurrence of renal infection from this source. Superficial ulceration of the colonic mucous membrane, not uncommon in constipation, favors the entrance of colon bacilli into the circulation.

In the treatment of infectious diseases, such as typhoid fever, pneumonia and empyema, the possibility of renal infection must be remembered, and efforts made to prevent its occurrence by the use of urinary antiseptics, such as hexamethylene-tetramin (urotropin) and the ingestion of as large quantities of water as possible, without taxing too much the water excreting power of the kidneys. In any condition where catheterization of the bladder becomes necessary, every possible precaution to secure asepsis must be made. If cystitis occurs, it should be actively treated as a source of great danger, and the use of ordinary antiseptics and the drinking freely of water are the important prophylactic measures to be followed in combating this complication. Catheterization of the ureters must not be undertaken lightly, and in the performance extreme care must be taken to avoid infection. A calculus in the kidney is always a source of danger. It may injure the contiguous renal structures, and so create a local point of diminished resistance that invites infection. It should, therefore, be removed.

When the kidney or its pelvis has been infected, an attempt is made to destroy the offending organisms and to raise the resistance of the patient. The administration of urinary antiseptics, of which hexamethylene-tetramin (urotropin), 5-grain (0.32 Gm.) doses, three or four times daily, is strongly indicated. In the majority of cases this drug is without harmful effect, but in a few cases hematuria and strangury occur. If these symptoms appear in the course of a pyogenic infection of the kidneys, the drug must be withdrawn for a few days in order to determine the origin of the symptoms. Should the hematuria be the result of the taking of hexamethylene-tetramin (urotropin), much harm will be done to the kidneys by its continuance.

It is probable that bacteria which will grow in an acid urine, will grow less well, or even die, in an alkaline medium. The converse may be said of bacteria growing well in alkaline urine. Therefore, the reaction of the urine should be changed.

Boric, benzoic, salicylic or camphoric acids should be given when the urine is alkaline; if acid, alkaline waters, sodium bicarbonate, potassium citrate or acetate should be used.

Diet is important, and must be arranged to suit the individual case. The patient must be adequately nourished in order to raise his resistance to the disease. Even though fever be present, the diet must be as generous as the digestion will tolerate. A strict milk diet is to be avoided. As a rule, a mixed diet, with a minimum of protein, 70 to 80 Gms. (2.2 to 2.5 $\frac{3}{4}$ ), is best. Alcohol should be avoided. Water should be drunk freely, the amount ingested depending upon the ability of the kidneys to excrete water.

As a rule the pain is not of sufficient severity to call for medication. Hot stupes or hot-water bottles, applied to the lumbar region, often relieve the aching. When pain becomes an annoying symptom, acetylsalicylic acid (aspirin), 5 to 10 grains (0.32 to 0.6 Gm.), or acetphenetidin, 5 to 10 grains (0.32 to 0.6 Gm.), may be employed. For severe pain it may be necessary to use codein or morphin.

When possible, a bacteriological study of the urine must be made, with a view of isolating the offending organism, from which to make a vaccine. If more than one micro-organism be present, a polyvalent vaccine should be made containing the various germs in their numerical relationships as found in the culture. About fifty million bacteria should be given as the first dose. The next dose should be increased or decreased, depending upon the presence or absence of a reaction and its severity. They should be administered hypodermically at intervals of from five to seven days. Stock vaccines should not be used.

Operation must be resorted to, if the constitutional symptoms are severe, and particularly if the patient shows increasing evidence of toxemia. It must, likewise, be resorted to in the absence of severe constitutional symptoms, if the patient be gradually losing weight and strength. Before operating it is imperative to catheterize the ureters to determine definitely whether one or both kidneys are diseased, and to study the functional activity of each kidney with the phenolsulphonephthalein test. Thus we are able to determine, not only which kidney is diseased, but, if both are



affected, which one the more seriously. With this knowledge it will not happen that the better of the two kidneys is removed, resulting in the speedy death of the patient. The removal of an infected kidney is frequently followed by a marked increase in the functional power of the remaining one.

### PERINEPHRITIC ABSCESS.

By this is meant suppuration in the connective tissue surrounding the kidneys. The abscess is practically always secondary to infection elsewhere, usually in the kidney or its pelvis. Rarely the source may be in some of the neighboring structures. A primary infection is conceivable, but this is so rare as to be negligible. In a series of cases studied by Braasch<sup>15</sup> the etiologic factors in the order of their frequency were (1) pyonephritis; (2) renal tuberculosis; (3) nephrolithiasis; (4) abscess of the renal cortex, and (5) traumatic rupture of the kidney.

The symptom of first importance is pain, sometimes increased by pressure, in the upper lateral aspect of the abdomen or in the lumbar region. Chills, fever and sweats, or chilliness, with fever of the continued type, occur. Edema of the skin over the affected area is observed in some of the cases. If the disease be of sufficiently long duration, emaciation eventually sets in. The leucocytes, particularly the polynuclears, are usually very much increased. When the abscess is the result of infection from the kidney, pus and blood may be found in the urine, sometimes in microscopic quantities only. As the pus cells and erythrocytes may be present intermittently, repeated examinations of the urine are necessary. Bacteriologic examination of the urine collected separately from each kidney often yields valuable information. A cystoscopic examination must be made and the urine from each kidney separately examined for abnormal constituents. A functional test of each kidney is essential, inasmuch as the results of such an inquiry help to determine the nature of the surgical treatment to be followed. For the same reason the kidney and its pelvis should be studied with the *x*-ray.

The treatment consists in evacuating the pus. Whether the abscess be simply drained or a nephrectomy performed is

dependent on the state of the kidney as diagnosed by a study of the urine, in conjunction with the *x*-ray studies. If the kidney be tuberculous or a pyonephrosis be present, the organ should be removed, provided that the opposite kidney is functioning properly. If the source of the pus consists of a superficial abscess in the renal cortex, simple drainage of the perirenal abscess will usually effect a cure.

### TUBERCULOSIS OF THE KIDNEY.

The kidney may be affected by tuberculosis to the almost complete exclusion of other parts of the body, or the renal lesion may be merely part and parcel of a general miliary tuberculosis. In the latter event the diagnosis is made with difficulty, if at all, and there is no treatment directed to the renal infection that is of any value. The renal disease is but a detail in the general picture.

The kidney is probably never the seat of primary tuberculosis. In almost every instance the infection is carried to the kidney from a focus elsewhere in the body, the primary site of which may be a slight infection at the apex of a lung, or a diseased peribronchial, or a mediastinal or mesenteric lymph gland. Infection may occur from the bladder or other parts of the genito-urinary system (urogenous or ascending infection), but this is probably very much less common than hematogenous infection. Very rarely infection may occur by contiguity, as from the adrenal, or the intestine. As a rule, but one kidney is affected, the right probably more often than the left. The disease may occur at any age, but is most common between the ages of 20 and 40 years.

The pathologic changes that take place in the kidney are the same as those that occur in any other part of the body infected with tubercle bacilli. Small grayish tubercles appear, their location depending upon the point where the first invaders lodge. The primary tubercles may, therefore, be found in the neighborhood of the Malpighian bodies, in the uriniferous tubules, in the pyramids, or in the papillæ. Usually the infection occurs in the pyramids. The minute tubercles grow larger, caseate, often coalescing to form large necrotic areas which ulcerate into the renal pelvis and discharge the pus into the urine, leaving large cavities that

extend deeply into the renal substance. Secondly, in such cases, the renal pelvis, ureter, bladder, and other parts of the genito-urinary system become infected. The kidney may remain about normal in size, unless occlusion of the ureter takes place, in which event pyonephrosis develops and the kidney may become a large sac filled with caseous material. The occlusion of the ureter may result in a disappearance of pus from the urine and add to the difficulty of diagnosis of cases coming under observation for the first time after the occlusion has occurred. A cystoscopic examination will aid very greatly in making a diagnosis. Late in the disease the surrounding tissue and the opposite kidney may become infected. The tuberculous abscess may extend through the renal substance, and, rupturing the capsule, may burrow outward and discharge upon the surface of the body, or may extend into the peritoneum, and, consequently, infect the entire abdominal cavity.

Unfortunately in the early stage of the disease, when an accurate diagnosis is of the utmost importance, the absence or vagueness of the symptomatology renders an unqualified diagnosis most difficult. Symptoms may be absent until late in the disease, in many cases; in others a very gradual loss of weight with malaise and slight weakness may be the only symptoms. The urinary changes are the earliest and most constant, and of these, the appearance of blood is the most important. The hemorrhage may be so slight as to be detected only by the microscope, but when found should always raise the question of tuberculosis. Later when caseation occurs, pus may appear in the urine microscopically, or macroscopically, and casts may occur. The macroscopic appearance of pus may be intermittent, although in many cases it is absent throughout the course of the disease. Sometimes tubercle bacilli may be found. Their absence from the sediment does not exclude the diagnosis of renal tuberculosis. The reaction of the urine is acid, unless an associated infection with a pyogenic organism occurs, in which case the urine is alkaline, and deposits, on standing, a heavy sediment of pus, mucus and phosphates. The quantity of urine in 24 hours is likely to be normal, though it may be either increased or decreased. Commonly patients com-

plain of frequent micturition at night. Pain may occur during the act of micturition, even when cystitis is absent.

A dull ache is sometimes felt in the lumbar regions, but as a rule it is slight. If a plug of caseous material or a blood-clot lodges in the ureter, severe colicky pains, as in nephrolithiasis, may occur. Fever may be absent until late in the disease, when the evidences of a marked sepsis, chills, fever and sweats may be a conspicuous clinical feature. As a rule, the temperature is subnormal during the day and at night ascends to 100° F. (37.7° C.), or slightly less. Night sweats may occur.

Physical examination may reveal tenderness on pressure in the lumbar region or loin. As a rule, no mass is felt, but when a pyonephrosis is present the kidney may be palpable, and is usually found to be very tender. The size of the palpable kidney may vary from time to time, depending upon the patency of the ureter. If pus has accumulated in the perirenal and pararenal tissues, edema in the loin or lumbar region may be present.

Because of the importance of early diagnosis and the untrustworthiness of general symptoms of renal tuberculosis, the finding of blood in the urine, either in microscopic or macroscopic amounts, calls for a thorough investigation, including a cystoscopic examination and catheterization of the ureters. Occasionally a whitish mass, cheese-like in consistency and moulded to the shape of the ureter, may be seen emerging from the mouth of the ureter by means of the cystoscope. The urine thus obtained should be painstakingly examined for tubercle bacilli, which, if found, are of extreme importance in the diagnosis, unless there is widespread tuberculosis in other parts of the body, in which event they commonly find their way into the urine without producing lesions that are recognizable by the naked eye. Catheterization of the ureter, filling the ureter, pelvis and calices of the kidney with thorium, followed by a radiographic or fluoroscopic examination, often aids the diagnosis. The cheese-like material may be injected into a guinea pig, so that the tuberculous character may be proven.

In the vast majority of instances surgical treatment is necessary. The operations which may be considered are

nephrectomy, nephrotomy, nephrostomy and partial resection of the kidney. Nephrectomy is the operation of choice. Statistics of various surgeons show a good number of cures. The mortality rate, which was formerly from 20 per cent. to 25 per cent., has been reduced to 10 per cent. The statistics of some clinics show the mortality rate to be as low as 3 per cent. Of nephrotomized cases, Brown<sup>16</sup> states that of a series of 72 cases from different surgical clinics, 7 were cured completely, 18 had a persistent fistula, 28 required a secondary nephrectomy, and 21 died. In this series, therefore, the mortality rate was extremely high, and is in marked contrast to the 10 per cent. rate in nephrectomy. Partial resection of the kidney is inadvisable, for the reason that some diseased tissue may be left behind to act as the focus for further trouble.

Before nephrectomy is done it is absolutely imperative to ascertain the health of the opposite kidney, and likewise its functional capacity. For this purpose the ureters must be catheterized, and the urine of the presumably sound kidney studied clinically and microscopically. The presence of a small amount of albumin unaccompanied by pus or blood is not a contraindication to operation. If the phenolsulphonaphthaleïn test repeatedly shows the functional capacity to be considerably impaired, a nephrectomy must not be undertaken. A slight impairment as shown by the test is not a contraindication to operation, as often, after operation, the function of the remaining kidney increases quite naturally. Marked retention in the blood of urea or of creatinin indicates serious renal inefficiency and contraindicates the operation. Surgery should not be attempted when there is extensive tuberculosis elsewhere in the body.

If the patient can be kept under close observation and control, and if the tuberculosis of the kidney is not extensive, an effort may be made to secure improvement by non-surgical measures. The effort, however, must not be prolonged. If definite improvement is not secured after two months' treatment, or if at the end of two weeks' treatment the patient is worse, even though but to a slight degree, surgical interference must not be postponed.



The non-surgical treatment, both before and after the operation, must be conducted along the lines generally adopted for tuberculosis elsewhere in the body. Of the measures employed the most important are the hygienic and dietetic. The skin must be kept in good condition, in order to maintain its function as an excretory organ at the highest level. For this a warm cleansing bath, using a bland soap, such as castile, should be taken at least three times a week, preferably at night just before retiring. If taken during the day the patient must remain indoors for an hour or two to avoid chilling. If the skin be dry and harsh, gentle massage with equal parts of cocoa butter and lanolin, with sufficient almond oil to make the mixture of the consistency of soft butter, is of value. The patient should be trained in the use of cold baths, which can best be done by ordering a sponge bath of tepid water each morning upon arising. The temperature of the water should be lowered a degree each morning or two until a cold bath is being taken. This bath should be followed by a brisk rub with a coarse Turkish towel.

Sunlight is very important. The patient should sit in the sun with the head covered by a hat or by a small sunshade. In cool or cold weather almost all the hours of sunlight may be utilized. In hot weather only certain hours in the early morning and in the evening may be used. Colored glasses may be worn to protect the eyes from the glare. As much time as is possible should be spent out-of-doors in a comfortable reclining chair on a veranda, sheltered from the wind. If the air be chilly or cold, woolen underwear should be worn and sufficient blankets used to keep out the cold. The danger of renal congestion from chilling of the body surface must always be remembered.

Patients who have recovered sufficiently from the immediate effects of the operation, and who can afford to do so, should seek a climate in which the spending of a maximum amount of time out-of-doors is possible. The climate best suited to these patients is to be found in southern California, Arizona, New Mexico, Utah, Colorado and Hawaii.

There are a number of factors to be considered in advising climatic treatment, one of the most important being the character of the food supply to be obtained at the resort

selected. Much better results will be secured at home, in a poor climate, with a properly arranged dietary of good foods, than can be possible in an ideal climate with improperly cooked foods of poor quality.

Rest, both mentally and physically, should be insisted upon in the beginning. All business cares, household worries, anxieties of every kind must be removed as far as possible. Most of the day should be spent in a recumbent or semi-recumbent posture, and ten consecutive hours should be spent in bed, *e.g.*, 9 P.M. to 7 A.M. The amount of exercise permitted varies with the strength of the patient and the effect upon the kidney. Fatigue is to be avoided always. The appearance of albuminuria, or, if present, its increase after exercise, is an evidence of harm. Very active exercise, such as tennis, rowing, running, climbing, must be prohibited. Walking, the distance increased as the patient's strength increases, is the best form of exercise. Late in convalescence, golf may be played, but no efforts in competition, such as match plays, must be undertaken. Before the patient is strong enough to exercise, the muscles may be kept in a semblance of condition by general massage.

The diet must be arranged in accordance with the functional capacity of the kidneys. The proteins in the diet should never exceed the needs of the normal adult, 80 to 100 Gms. (2.5 to 3.2 oz.), even when the number of calories per day is greatly increased. If uric acid, urea or creatinin retention in the blood rises, the amount of proteins must be reduced. For the average case a mixed diet of plain, easily digested foods should be arranged. A glass of milk, or milk and eggs flavored to suit the taste of the patient, may be taken between breakfast and lunch, and between lunch and dinner. The amount of fluid depends entirely upon the ability of the kidney to excrete water, the chief object being always to secure the elimination of 48 ounces (1419 mls) of urine daily in an adult.

The value of the use of tuberculin has not been conclusively demonstrated. Its use is not without danger, and in the present state of our knowledge it is inadvisable to recommend its use in general practice.

### TUMORS OF THE KIDNEY.

Tumors of the kidney are not very common, and often present great difficulties in diagnosis. Among the new growths that affect the kidney are hypernephroma, carcinoma, sarcoma and adenoma. The cardinal symptoms of renal tumor are hematuria, pain and tumor. When all these are present the neoplasm probably has grown to great size. New growths, when located at the upper pole of the kidney, may attain considerable size, and yet not be palpable. The pain is often absent and when present is extremely variable in its character, and not at all diagnostic. It may vary from a dull ache in the lumbar region to severe neuralgic pains along the course of the sacral and pelvic nerves. If blood-clots lodge in the ureter, pain similar to that experienced in nephrolithiasis occurs. As a rule, the hematuria is intermittent. It may be observed in but a few specimens of urine or may persist for days. The very sudden disappearance of blood from the urine may be due to the blockage of the ureter. As the different tumors of the kidney present no symptoms that point to their identity, it is usually impossible to differentiate them clinically. It is to be remembered that sarcoma of the kidney and tumors arising from the inclusion of embryonal tissue in the renal substance occur in early life. The other neoplasms are more likely to occur after the age of 30 years, the majority being found between the ages 40 and 60 years.

The most important function of the physician is the diagnosis of the presence of a renal tumor as early as possible, and in this attempt all cases of hematuria should be studied with care, and the real cause of the blood determined. It is not sufficient to surmise a cause. There is no medical treatment. In the light of our present knowledge, there is no drug or combination of drugs that will "cure" the patient of a renal tumor. When possible the tumor should be removed surgically.

### CONGENITAL CYSTIC DEGENERATION OF THE KIDNEYS.

This is a rare affection, probably due to developmental error. The kidneys become very greatly increased in size

and appear to be composed entirely of an aggregation of cysts of varying size. The growth of the renal cysts in the fetus may be so great as to prevent its passage through the birth canal. The cysts may not be large at birth, but later gradually increase in size, so that the patient dies in adult life because of renal insufficiency. Both kidneys are usually affected, and cysts may also be found in the liver. Treatment is of no avail, the patient dying eventually of uremia.

## BIBLIOGRAPHY.

1. Sahli: Diagnostic Methods, German Ed. 4, English Trans., Philadelphia, 1905.
2. *Ibid.*
3. Die Erkrankungen der Nieren, Berlin, 1902; also Nothnagel's Encyclopedia of Practical Medicine, American Ed., Philadelphia, 1905.
4. MacCallum, W. G.: A Textbook of Pathology, Philadelphia, 1916.
5. A Fatal Case of Poisoning by Bichloride of Mercury, Bull. Med.-Chir. College, Philadelphia, 1914, x, 5.
6. Billings: Forchheimer's Therapeutics of Internal Diseases, New York and London, iv, 40.
7. Jour. Pharmacol. and Exper. Therap., 1910, i, 579.
8. Arch. Int. Med., 1915, xvi, 733.
9. Jour. Am. Med. Assn., 1916, lxvii, 929.
10. Arch. Int. Med., 1916, xvii, 570.
11. Jour. Am. Med. Assn., 1915, lxv, 1234.
12. Collected Papers by the Staff of St. Mary's Hospital, Mayo Clinic, 1905-1909, Philadelphia, 1911.
13. Jour. Am. Med. Assn., 1915, lxv, 1234.
14. Mayo, W. J., Braasch, W. F., and McCarty, W. C.: Relation of Anomalous Blood-vessels to Hydronephrosis, Jour. Am. Med. Assn., 1909, iii, 1383.
15. Surg., Gynec., and Obstet., 1915, xxi, 631.
16. Osler, William: Modern Medicine, 1909, vi, 296.

# Diseases of the Digestive System

BY

B. B. VINCENT LYON, A.B., M.D.,

Chief of Clinic, Gastro-enterological Department, Jefferson Hospital;  
Pathologist, Methodist Episcopal Hospital, Philadelphia;

AND

LOUIS LEHRFELD, A.M., M.D.,

Editor of the "Monthly Bulletin" and Agent for the Prevention of  
Disease Department of Public Health and Charities, Philadelphia;  
Secretary, Pneumonia Commission, Philadelphia; formerly Assistant  
Surgeon United States Navy.





# Diseases of the Digestive System.

---

## FOREWORD.

IN the section on Diseases of the Digestive System, effort has been made to detail such treatment as may be within the reach of the average practitioner. A brief description of the pathology and etiology has also been given in order to make the principles of treatment more comprehensive.

Special stress has been laid on the relation of mouth infections to general systemic diseases. Heretofore the average medical practitioner has seemed to overlook the foci of infection about the teeth, tonsils, and sinuses of the skull, which are now known to be actively engaged in the dissemination of organic diseases.

Special attention is also given to the subject of acute infectious jaundice, which is a trench disease common among the troops throughout Europe. This infection is of great importance to the Military, Federal, and State health officials, and has therefore been reproduced in this section by permission of the Surgeon-General of the Public Health Service, who has recently published the latest information on this subject matter.

## DISEASES OF THE MOUTH.

### DISEASES OF THE MOUTH AS RELATED TO GENERAL SYSTEMIC DISEASES.

It is only within recent years that infections of the mouth have been found to be related to diseases of the heart, joints, muscles, nerves, stomach, and vascular system. These infections may occur about the teeth, in the gums, in the tonsils, and in the various bony sinuses of the skull which are directly communicating with the mouth. The anatomic structure of the mouth is such that it possesses fossæ, depressions, crypts,

and indentations, which may become hiding places for the fertile development of the various pathogenic micro-organisms. Here they lie dormant until the body resistance at some time or other is lowered, or, in symbiosis with other micro-organisms, they may be hastened into activity, and become disseminated through the lymph channels or by the blood to other tissues in the body for which they have special affinity, and there produce definite lesions.

It is now known that certain strains of pathogenic bacteria have the special qualification of selecting definite parts of the body for their growth and development. For instance, it has been demonstrated that certain strains of streptococci show special affinity for joints, tendons, muscles, and valves of the heart. Under certain other conditions the same streptococci, possessing varying qualities in virulence or high specificity, may show preference for the gall-bladder, the appendix, the stomach, or the duodenum. Inoculations of these germs into experimental animals have produced lesions in the same organs from which they had been originally taken. This remarkable discovery has given new light and impetus along the lines of biologic therapy. In the manufacture of vaccines for the treatment of these various infections it is necessary not only to isolate the micro-organism concerned, but that particular type of germ which has special selection for diseased areas.

Commercialized dentistry has opened the way for the faulty mechanical dental work responsible for the large proportion of mouth infections which the medical profession must deal with to-day. Imperfectly adjusted cappings, bridge work, and fillings may readily favor breeding places for pathogenic micro-organisms. Haphazard dental work performed by various commercial dentists has given the medical profession an entirely new chain of diseases to deal with, namely, septic infections in the mouth. Undoubtedly the increased attention given by the public to the care of teeth has decreased the percentage of digestive disorders, but the careless correction of defects in the mouth has given rise to focal infections which may even surpass in importance the original defects in the teeth.

Teeth devitalized in the course of treatment by the dentist

are very susceptible to infecting agents, and, being deprived of the nerve supply, inflammatory conditions may occur, even without pain, and thus be overlooked by the dentist and physician; focal pockets of purulent material may accumulate under crowns, beneath bridge work, or at the roots of devitalized teeth, and give no local symptoms. Absorption, however, of toxic material and invasion of the body by germs generated at these pus pockets about the teeth may produce such diseases as acute articular rheumatism, chronic arthritis, arthritis deformans, pericarditis, endocarditis, and myocarditis. A whole chain of other diseases also may originate from these teeth infections, such as furunculosis, myalgia, anemia, nephritis, gastritis, and enteritis. Many cases of ill-defined diseases, such as neurasthenia, melancholia, physical and mental depression, may have their origin in purulent infections in or about diseased teeth.

The tonsils also may be the seat of hidden pus pockets, acting as distributing foci of micro-organisms lodged in structures distant from the original seat of infection. Thus, acute articular rheumatism, pericarditis, and endocarditis are often traced to recurrent attacks of sore throat.

The mouth also is subject to invasion by pathogenic micro-organisms, because of its intimate connections with the middle ear, the mastoid sinuses, the ethmoid, sphenoid and maxillary sinuses, the larynx, the esophagus, and the lymphatics of the neck. It is not difficult to understand that these various sinuses and organs either may become infected through the mouth or that the mouth may be the secondary area of infection subsequent to the primary invasion of the organs just named.

Various micro-organisms may be found in the mouth of the healthy, normal individual. The salivary secretions with their enzymes are sufficient in most instances to stay the activity of these germs. When, however, the bodily resistance is lowered through disease, local or systemic, the bacteria in the mouth may find conditions favorable for development and invasion. It is well known that persons may carry pathogenic bacteria in secretions of the nose and throat, and that they themselves may not suffer from their presence. Thus the streptococcus, the diphtheria bacillus, the pneumococcus, the

influenza bacillus, and the meningococcus have been isolated in secretions of normal, healthy individuals. It is only when traumatism, injury, or low resistance of the patient exists that these germs invade the body, with more or less serious results.

Daland<sup>1</sup> in discussing oral sepsis makes the following conclusions on this subject:

1. Focal infection is one of the causes of acute and chronic arthritis, periarthritis, arthritis deformans, ostitis, endocarditis, pericarditis, or myocarditis, endarteritis, phlebitis, acute and chronic parenchymatous nephritis, cholecystitis, cholelithiasis, gastric and duodenal ulcer, appendicitis, meningitis, thyroiditis, neuritis, oöphoritis, cerebritis, myelitis, ocular diseases and furunculosis, and is the *unrecognized* cause of other diseases.

2. The results of focal infection are due to the variety and varying virulence of the micro-organisms, the duration of the focus, the quantity of micro-organisms and toxins entering the circulation, the rapidity of absorption, the integrity of the tissues, and the susceptibility or immunity of the patient. The virulence of the micro-organism, rather than the size of the lesion, is important. Although the exact effects of toxemia are not fully understood, it plays an important rôle in focal infection.

3. The usual location of focal infection is in the head cavities, the order of frequency being the mouth, the tonsils, and the sinuses. The diagnosis of chronic focal infection is sometimes easy, but more often it is difficult. A common error is to recognize but one focus when more than one exists, and this is especially true of the teeth.

Freudenberger,<sup>2</sup> in commenting on the relation of the teeth to tuberculosis, in a study of 297 cases, claims that it is a question whether tuberculous disease and mixed infection among his patients arose through the condition of their teeth and gums, or whether their tuberculous condition affected their teeth and gums secondarily. It is assumed, at any rate, that a bad condition of the teeth and gums offers a good medium for the growth of the tubercle bacillus, which has often been found in teeth containing cavities. Sore teeth and tender gums, however, may be the cause of insufficient and ineffectual mastication.



## TREATMENT.

Affections of the joints, indefinite, vague, general symptoms, and cardiac disease usually call for inspection of the mouth. Very often no local lesion is visible. Palpation of the gums and teeth may locate areas of tenderness. The tonsils may be perfectly clean on the surface, free from accumulations of cryptic secretions, and present no evidence of disease, yet beneath the surface there may be distinct definite pus pockets. The history of repeated attacks of tonsillitis may lead to the suspicion of hidden areas of infection. Each tooth should be tapped by a metal instrument to determine the presence or absence of pain. It may be necessary to have the consultation of a dentist for the determination of carious or diseased teeth. Even after all this inspection, there may be no visible evidence of disease. If the suspicion, however, is strong that infection exists about the roots of the teeth, an *x*-ray examination is in order. Roentgenologists state that in some instances the greatest detail in the taking of pictures often fails to show pus pockets, which do not throw shadows of diseased roots.

Every case of infection about the teeth and gums should be referred to a competent dentist. The usual application of tincture of iodine by the medical practitioner is not sufficient. Pus pockets should be opened and drained just as similar surgical conditions in other parts of the body.

There has been a common tendency on the part of physicians to recommend the extraction of teeth where diseased gums and purulent infection exist. This haphazard recommendation is condemned on the grounds that pus pockets may be opened, curetted, and successfully drained, without necessarily requiring the removal of teeth. This does not hold true, however, in all cases, such as in pyorrhea of long standing, when extraction gives more speedy results and an early abatement of symptoms. I have in mind a special case of cardio-renal disease in which medication accomplished no results until all of the teeth, both upper and lower, in a patient of middle age, were extracted. In this instance pyorrhea alveolaris, carious teeth, and ulcerative stomatitis were present.

As a general mouth-wash, liquor antisepticus alkalinus is recommended in mouth infections. Listerine and peroxid in

equal parts are also valuable. In the absence of these, potassium permanganate in solution (1:500) may also be used.

Diseased tonsils call for early removal. Repeated attacks of tonsillitis indicate that there is a focal infection present, which is lighted up from time to time. It is advisable that the tonsils be removed during an interval of these attacks. Partial tonsillectomies, in which stumps of tissue remain between the pillars of the fauces, may continue to act as foci of infection. It is, therefore, advisable that diseased tonsils be removed completely. I have frequently met with cases of tonsillitis in patients who claimed that their tonsils were removed in childhood. Hypertrophy of the stumps may take place, and become subject to invasion as in the original tonsil.

Sinusitis and middle ear disease very often have their origin of infection in the mouth. For the treatment of these conditions appropriate textbooks on these subjects should be consulted.

Infection of the sinuses of the skull is very difficult to treat by local measures. Mixed vaccines are recommended. When these infections become purulent, however, surgical intervention is required.

### **FISSURES OF THE LIPS.** (Rhagades of Commissures.)

This condition is characterized by a fissured eczema-like maceration of the corners of the mouth, which is quite painful when the lips are parted, or when fluids come in contact with the affected parts. It occurs most frequently in children, but may also occur in adults whose physical condition is below par. The angles of the mouth are reddened and scale-like, and the epithelium is deeply fissured. Itching and burning are characteristic symptoms. Both corners of the mouth are usually affected at the same time.

The disease seems to bear some relation to the home environments of those affected, it being found most frequently among those living in poor surroundings and under poor discipline, of personal hygiene. In adults it is generally associated with chronic diseases, and is very resistant to treatment.

Fissures should be cauterized with a solution of silver nitrate, 10 grains to the ounce (0.65 Gms. to 30 mls), followed by the application of an ointment, the composition of which is as follows:

℞ Hydrarg. oxid. flav. .... gr. ss (0.032 Gm.).

Petrolatum album ..... 3j (3.90 Gms.).

S.: Apply locally at bedtime.

Zinc ointment may also be applied with beneficial effect. Camphor ice is also recommended. The general health of the patient should be given careful consideration and treatment instituted according to the individual requirements.

## HERPES LABIALIS.

### (Cold Sores; Fever Blisters.)

This condition is characterized by the eruption of vesicles varying in size from a pin-head to that of a split pea upon the exposed surface of the lips and skin adjacent thereto. These vesicles may be single, multiple, or confluent, and rest upon hyperemic bases. Their appearance is usually preceded by a burning or tingling sensation of the lips, soon followed by an eruption of varying sized watery blisters. The lips are dry, glazed, and reddened. The contents of the vesicles are at first clear, but soon become milky. Absorption takes place after several days, leaving reddish brown crusts, which soon fall off. Picking at these crusts, however, will cause them to bleed, leaving denuded painful surfaces. In children there is a tendency to pick at these crustations, causing irritation and inflammation in the tissues immediately adjacent.

Herpes occurs in digestive disorders, constipation, febrile diseases, pneumonia, malaria, and in acute catarrhal affections of the nose and throat.

*Treatment.* It is very difficult to abort eruptions of herpes because they appear so rapidly. Sweet spirits of nitre containing a few drops of tincture of belladonna; or spirits of camphor may be used to hasten the drying of the vesicles. Alcohol or tincture of benzoin is also recommended. After the scabs have formed, they should be softened with cold cream, yellow oxid ointment, or white vaselin.

### FOUL BREATH.

A continuously offensive breath is always indicative of an abnormal condition. After eating certain foods, the breath may have an odor characteristic of these substances, such as occurs after the ingestion of alcohol, creosote, mint, onions, and garlic. A disagreeable odor from the mouth which persists may signify the presence of decayed teeth, diseased tonsils, inflammatory disease of the nose, throat, and sinuses of the face, gastro-intestinal disorders, chronic constipation, Bright's disease, diabetes, fetid bronchitis, gangrene of the lungs, pulmonary tuberculosis, cancrum oris, cancer of the tongue or larynx, or may be associated with chronic wasting diseases. An odor may also be imparted to the breath following poisoning by mercury, arsenic, lead, and phosphorus. The constant use of bromids and iodids may also give a characteristic odor to the breath.

The *treatment* of this condition of course deals with the removal of the cause. Attention to the teeth is very important and the occasional administration of saline purges is indicated. The mouth should be washed after each meal with a solution of peroxid and listerine equal parts, or with Dobell's solution or liquor antisepticus alkalinus.

### GINGIVITIS.

Inflammatory conditions of the gums may involve the superficial surface or marginal areas close up to the teeth or may extend to the deeper structures as in alveolar processes of the maxillary bones.

Retained particles of putrefactive and decomposed food or general systemic diseases may bring about a superficial gingivitis. Irritation from overhanging crowns and faulty fillings may also bring about this condition. In certain diseases, such as tuberculosis, syphilis, Bright's disease, scurvy, diabetes, in cases of alcoholism, in advanced grades of anemia, and sometimes in febrile diseases, the gums are hyperemic, tender, and bleed easily on touch. Burns by chemicals, such as bichlorid of mercury, carbolic acid, and other corrosive medications, may cause inflammatory conditions of the gum. The internal

administration of mercury and iodids may also cause gingivitis. Various industrial occupations in lead, phosphorus, and mercury may affect the gums. The causative factor must be removed. Imperfect and faultily done work should be corrected, and industrial pursuits should be either abandoned or controlled by careful medical supervision. Systemic diseases should be treated accordingly.

A suitable mouth wash for superficial gingivitis is as follows:

Sod. bicarbonatis,	
Sod. biboratis .....	āā 3j (3.90 Gms.).
Acidi carbolici .....	3ss (1.95 Gms.).
Glycerini .....	f3j (31 Gms.).
Aqua destillata, q. s. ad .....	f3iv (124.40 Gms.).
M. S.: One tablespoonful (15 mls) to a half-glass of water and use as a mouth-wash every two hours.	

### PYORRHEA ALVEOLARIS.

This is an infection of the mouth characterized by a bleeding, spongy, purulent condition of the gums and by inflammation of the pericemental membrane of the teeth. This may arise from local disease caused by infection from carious teeth with the various pathogenic organisms—bacilli, cocci, or amebæ. Crustations of tartar at the neck of the teeth impinging upon the gums may be the origin of this infection, or it may begin in uric acid deposits at the roots of the teeth in the case of certain constitutional diseases, such as gout and lithic diatheses.

"Two decades find us almost exactly where we were when I started to practice dentistry twenty-three years ago," says Price,<sup>3</sup> of Cleveland, at the 67th annual session of the American Medical Association, Detroit, June, 1916. "We cannot come together and express a common idea on the etiology of pyorrhea, and be noted in supporting and substantiating it." This is the general opinion expressed by some of the most prominent American dental authorities to-day. Only recently a well-known research worker proclaimed that the *Endameba buccalis* was the cause of every case of pyorrhea, and that this disease was present among 95 per cent. of the population. This statement, however, has been disproved many times by



various bacteriological experiments conducted in research laboratories. Among the organisms which have been isolated from the diseased gums and pyorrhea are the streptococcus, pneumococcus, staphylococcus, staphylococcus aureus, micrococcus catarrhalis, saccharomyces, treponema mucosum, influenza bacillus, diphtheroids, and endameba. The variety of bacteria found indicates that one or more causative agents may singly or jointly produce the same pathological condition known as pyorrhea. It appears, however, that the streptococcus is the principal and most frequent active cause of the disease. Recent investigations of the characteristics of the streptococcus point to the fact that this micro-organism has many possibilities, and it varies in virulence according to the strain or family group from which it is derived, and according to the resisting power of the patient. This bacterium, under varying conditions of growth, may possess different characteristics, being capable of various changes, and even convertible into micro-organisms resembling the pneumococcus, which in turn may be reconverted into the streptococcus. These micro-organisms, depending upon their strain and characteristics, possess selective action for certain parts of the body, showing preference to the joints, the heart, the kidneys, and other organs. When recovered from these various parts of the body and injected into laboratory animals, they produce exactly the same lesions and in the same organs from which they are derived. These specific qualifications of various strains of the same germ bear an important relation to the treatment, since special strains of germs must be used in vaccine therapy depending upon their selective preference for certain parts of the body.

In view of the varied opinions expressed as to the cause of pyorrhea, it is safe to state that this disease is an infection of the gums, occurring most frequently in persons whose physical condition is below par. Rhein<sup>4</sup> states that infection is impossible in an individual otherwise physically well. "Pyorrhea alveolaris is the result of malnutrition plus infection, and also most frequently plus irritation." Talbot<sup>5</sup> claims that deposits on the roots of the teeth other than tartar are the detritus of destroyed bone, and are not deposits from the blood as was formerly supposed. Many authorities claim that

these deposits are composed of uric acid, which is precipitated from the blood. Talbot sums up the etiology of these diseases, as follows:

1. We have to contend with the bone (teeth) as an end organ in which the blood accumulates.
2. Stasis of the blood cuts off nutrition.
3. Local and constitutional irritation and chemical changes of the blood set up a low form of inflammation and bone absorption.
4. The absorption is enhanced because of the transitory nature of the alveolar process.
5. Nerve end degeneration and arteriosclerosis occur.
6. Want of vital resistance assists absorption.

This disease is of extreme importance in that it may complicate general and infectious diseases, or that it may be the original focus for the dissemination of lesions in distant organs. Whether the disease arises in the cementum or in the periodontal membrane means little to the average practitioner, but it is of great concern to him whether the infection is a primary disease of the gums or whether it is a superadded infection occurring in the course of general systemic diseases.

Diseased conditions of the gum which are not readily amenable to treatment may have a cryptic source of infection about the root of one or more teeth. These so-called periapical abscesses may exist with few or no symptoms referable to the mouth. There may, however, be general symptoms of toxemia, of physical and mental depression, of malnutrition, anemia, and neurasthenia. Indefinite pains in the joints or definite heart lesions may have their origin in a purulent infection about the roots of the teeth. There is now a general tendency on the part of the medical profession to make a routine inspection of the mouth for the purpose of detecting hidden lesions about the teeth. The x-ray has been of invaluable service in this respect. Potter<sup>6</sup> recommends that a general survey of the denture by a series of dental films is an important adjunct to the examination of a pyorrhea case. It is often a short cut to a diagnosis, and is less disagreeable than an instrumental examination, but should supplement rather than displace other diagnostic methods. The most important findings are observed in the region of the intimate

bony investments of the roots, and are obtained only from the most critical röntgengrams. Radiographs do not always show looseness of the teeth, which is quite common in pyorrhea; and, on the other hand, abscesses are noted even when the teeth are intact and firm. Inasmuch as the teeth may be normal, and abscesses treated without interference with the function of the adjacent teeth, it is advisable that teeth be extracted only after consultation with the dentist. It is believed that heretofore physicians have ordered the extraction of teeth indiscriminately on the finding of periapical abscesses.

Bleeding gums is one of the first symptoms of pyorrhea. Painful mastication, toothache, neuralgia, foul-smelling breath, and digestive disorders are present. Palpation of the gums elicits tenderness. Their color varies from the bright beefy red to a dull brown, the latter being characteristic of the shrunken tissues below the neck of the teeth. Ulcerations of the gum and the mucous membrane also may occur.

### TREATMENT.

The diseased gum, with festoons hanging down between the crowns of the teeth, should be cut out by a sharp scalpel down to the alveolar border. Bleeding is encouraged. The gums may be scarified and rubbed with a soft brush. Tincture of iodine should be applied before and after scarification. A suitable gum-wash used in connection with the brush and massage advocated by Whistler, of Cleveland, is as follows:

Zinc sulpho-carbolate .....	3j (3.90 Gms.).
Alcohol .....	f3j (31 Gms.).
Aqua dist. ....	f3ij (62 Gms.).
Ol. mentha. pip. ....	m viij (0.50 mls).
M. S.: Use as a gum-wash.	

In looking over the recent literature on the treatment of pyorrhea, it is surprising to learn that very little is now said regarding the use of emetin hydrochlorid and ipecac, which only lately gained much popularity among the medical profession. Since these drugs are believed to be specific in combating the growth of the endameba, and since the micro-organism just named seems to be a complicating factor rather than the original cause of the disease, these drugs no longer possess an important part in the treatment.

General measures of treatment are indicated in pyorrhea. Saline purgatives of Epsom salt, citrate of magnesia, Rochelle salt, and other measures may be used. The salt action of these purgatives tends to relieve the blood system of its toxic products, and clears the intestinal tract of putrefactive material which would otherwise exaggerate the constitutional symptoms of pyorrhea. The internal administration of salicylates is also of great value in combating the micro-organisms and their products, absorbed from focal infections in the mouth, and distributed through the blood vascular system. Perversion of taste, which very often accompanies pyorrhea, may be overcome by using a mouth-wash of Dobell's solution, or a solution of potassium permanganate (1:500). Candy lozenges containing menthol, licorice, or peppermint are recommended when there is much fetor of the breath accompanying the disease.

General tonics, such as iron, nux vomica, and bichlorid of mercury, should be administered.

## CATARRHAL STOMATITIS.

Catarrhal stomatitis is a general inflammatory condition of the mucous membrane of the mouth attended with an increased secretion of saliva. It may arise from decomposing food particles, carious teeth, and from general uncleanness of the mouth. The constant chewing of tobacco, indulgence in strong alcoholic liquors, ingestion of highly spiced foods, and various drugs (iodids, arsenic, and bromids) may be the exciting causes. Constitutional diseases, infectious fevers, and certain occupations among chemicals, may be responsible for catarrhal inflammation of the mouth. Gastro-intestinal disorders, organic diseases, pregnancy, and lactation also may predispose to this disease.

It occurs not only in adults, but also frequently among children. Unhygienic surroundings, poor feeding, malnutrition, and gastro-intestinal disorders in the infant may act as exciting causes. The so-called pacifiers for the young infant may readily carry into the mouth infectious bacteria, resulting in ulceration and catarrhal inflammation. Throughout

the mouth may be found inflammatory glazed areas of mucous membrane, and here and there white patches.

The *treatment* is confined to general measures of cleanliness. In children the mouth can be cleansed by sweeping it with a finger or with a gauze-covered finger moistened with boric acid solution. The infant's mouth should be washed in this way before and after each feeding. The mother's nipple should also be washed with boric acid solution before and after each feeding. If the child uses the bottle, the nipple should be thoroughly boiled. In adults a suitable wash should be used several times during the day, and the teeth carefully brushed. Ulcerated areas may be treated by the application of a 10 per cent. solution of silver nitrate. Among the mouth-washes recommended are Dobell's solution, liquor antisepticus alkalinus, potassium permanganate (1:1000), and peroxid and glycerin (1:4) diluted with equal parts of water.

### APHTHOUS STOMATITIS.

(Herpetic, Vesicular, Follicular.)

This disease is manifested by a vesicular eruption on the mucous membrane of the mouth, cheek, tongue, or lips. These vesicles are the size of a pinhead, or slightly larger, and readily ulcerated, exhibiting a necrotic yellowish white base surrounded by a red areola. It occurs chiefly among children between the ages of six months and the end of the first dentition. Adults also may be affected. It is believed that the disease is caused by some toxic product affecting the nerves of the mouth, resulting in herpetic eruptions. Various bacteria have been isolated, but none other than those found normally in the secretions of the mouth. Among the predisposing causes are malnutrition, tuberculosis, diarrhea and enteritis, infectious fevers, and dentition. Lack of cleanliness, the use of stale and unclean milk, and the indiscriminate use of baby comforters are among the exciting factors which favor this disease. In adults it results from inattention to personal hygiene; it may be caused by carious teeth, or by the ingestion of highly spiced foods, or it may occur during the course of gastro-intestinal disorders. It may also occur



during the puerperium and lactation. When the vesicles break down and ulcerate, they become very painful and tender, especially during mastication, when the food comes in contact with the denuded areas. The vesicles are usually found inside the lower lip near the frenum on the mucous membrane of the cheeks near the back teeth, and along the edges of the tongue. Increased salivation usually accompanies the eruption. In children it may be associated with a slight rise of temperature, or it may precede an attack of gastro-enteritis.

Follicular stomatitis occurs during the course of childhood diseases—pneumonia or typhoid fever. The ulcers may coalesce, forming large necrotic areas, which are very painful. The disease usually runs a short course.

### TREATMENT.

In children every effort should be made to keep the mouth clean by swabbing it out thoroughly with a gauze-tipped finger moistened with boric acid solution, or with sodium bicarbonate, 10 grains to the ounce (0.65 Gm. to 30 mils) of water. Painful ulcers should be touched with a 2 per cent. solution of cocain before feeding, and should be cauterized by the application of a 10 per cent. solution of silver nitrate on a cotton-tipped applicator, or a saturated solution of iodoform in ether. The following internal medication is recommended by Anders:<sup>7</sup>

Potassii chloratis ..... gr. xxiv (1.55 Gms.).

Tr. of myrrhæ ..... gtt. x (0.60 mils).

Syr. acaciæ ..... fʒij (62 Gms.).

Aqua dest., q. s. ad ..... fʒiij (93 Gms.).

M. S.: fʒj (3.75 mils) every three hours for a child  
3 years of age.

A word of caution may be said against the use of the pacifier, playtoys, and rattles, which are frequently taken into the mouth by the infant. We have too often seen the mother pick up the pacifier or rattle from the floor, wipe it on the apron, and hand it to the child. Infection may be readily carried into the mouth by these toys. The pacifier should be strictly forbidden.

### BEDNAR'S APHTHÆ.

This is the name applied to an ulcerative condition of the soft palate and roof of the mouth among infants soon after birth, and is said to be caused by abrasions of the mucous membrane occasioned by the rough swabbing of the mouth by the nurse or doctor, or as the result of traumatism caused by friction of a long rubber nipple.

This condition is treated by swabbing the mouth with a solution of boric acid, and in more severe cases by the application of a 10 per cent. silver nitrate solution.

### THRUSH.

(Stomatitis Hyphomycetica; Parasitic or Mycotic Stomatitis.)

Infection of the mucous membrane of the mouth by a mycotic moldy growth resembling curds of milk, which are difficult to remove, may occur among infants, children, and the aged. This is known as *thrush*. It affects the lips, tongue, cheeks, soft palate, and occasionally the pharynx, esophagus, and upper respiratory passages. This mycelial or moldy growth is caused by one of the higher forms of bacteria known as the endomyces albicans, and produces a lesion in flake-like patches superimposed upon an already inflamed mucous membrane. The endomyces grows best in an acid medium. This fact is of therapeutic importance in that alkaline lotions are required to hinder its development. The disease occurs among nursing infants affected by gastrointestinal disorders, under-nourishment, tuberculosis, and other wasting diseases. Infection may be introduced into the mouth by unclean nipples, by table utensils, or by pacifiers and other trinkets. Older children may become infected by placing foreign bodies in the mouth. Among the aged the disease occurs in the course of cachetic diseases.

### TREATMENT.

The mouth of the infant should always be kept clean by cleansing it after each feeding with a gauze-tipped finger moistened in boric acid solution. The mother's breast should

be treated likewise before and after each feeding. Bottle-fed babies should receive generous care in the preparation of their feedings, and attention paid to the cleanliness of the nipples. The milk should be fresh, and free from contamination. Children should be taught not to place toys and other foreign bodies in their mouths, and parents should prohibit the use of pacifiers. In adults, and especially among the aged, the routine use of one of the common mouth-washes is advocated.

When the disease has developed, the affected parts should be treated with a solution of sodium bicarbonate or sodium biborate, 10 grains to the ounce (0.65 Gm. to 30 mils), on gauze-tipped finger or on a cotton-tipped applicator. Adults may use to advantage a mouth gargle of liquor antisepticus alkalinus, sodium bicarbonate, or sodium biborate, 10 grains to the ounce (0.65 Gm. to 30 mils). The mycotic growth should not be removed, since it usually carries with it the mucous membrane, leaving ulcerated areas. Should ulcerations result, however, they should be treated by the application of silver nitrate, 10 grains to the ounce (0.65 Gm. to 3 mils).

### ULCERATIVE STOMATITIS.

(Vincent's Angina; Fetid Stomatitis; Putrid Sore Throat.)

This is an acute inflammatory and ulcerative infection of the gums, and later may extend to the maxillary bones. It occurs among children from the age of three to puberty, and also may affect adults. Unhygienic surroundings, together with poor feeding, under-nourishment and undermined physical health are predisposing factors. It may develop during the course of febrile diseases, measles, scarlet fever, typhoid, and other infections, or it may result from the invasion of bacteria into tissues whose resistance is lowered by chemical or mechanical irritation. Mercury, phosphorus, arsenic, iodids, and bromids may produce ulcerative stomatitis. It may occur in the course of syphilis, tuberculosis, cancer, and other wasting diseases.

The gums of the lower jaw become painful, reddened and swollen, and soon ulcerate, with dirty white necrotic areas

surrounded by bleeding, spongy tissue. If untreated, these ulcerations extend rapidly to other parts of the mouth, affecting the gums on the labial and lingual surfaces. The sloughing becomes discolored, and there is distinct fetor of the breath. There is an increased flow of saliva, which in cases of debility overflows, running down the corners of the mouth, producing redness and excoriations upon the skin of the lower jaw. In many instances the inflammatory condition actually may cause a periostitis of the maxillary bones, attended with pain and swelling of the sub-maxillary glands. The disease may be associated with fever, gastro-intestinal disorders, and extreme prostration. It may progress rapidly, causing extensive ulcerations of the mouth within from three to five days. In institutions this affection may become epidemic through the use of common eating utensils, cups, glasses, and towels.

#### TREATMENT.

The treatment calls for the adoption of sanitary measures in institutions where the disease occurs. The common use of eating utensils, glasses, and drinking-cups should be prohibited. Housing conditions should be improved. Patients should be encouraged to be out-doors in favorable weather. A mixed and wholesome diet should be given the inmates of institutions to avoid malnutrition, which in turn predisposes to infections. Ulcerative stomatitis should be treated by the use of a mouth-wash composed of 10 grains to the ounce (0.65 Gm. to 30 mils) solutions of sodium bicarbonate, sodium biborate, potassium chlorate, alum, or tannic acid, or potassium permanganate (1:500) may serve best.

The ulcerated area should be treated with peroxid of hydrogen, followed by the application of tincture of iodine or 10 per cent. solution of silver nitrate. Children may be given potassium chlorate internally, as described under Aphthous Stomatitis. (See p. 657.) This drug may also be given to adults, but is contraindicated in cases associated with nephritis. Under constant treatment the disease may be abated in from four days to one week. In cases of longer standing, the deeper structures become affected, resulting in alveolar necrosis. In such cases the necrotic areas should be curetted and

dusted with powdered iodoform once daily. The teeth, however, should not be removed unless carious.

The general constitutional condition of the patient should be improved by dietary measures—fresh fruits, vegetables, fresh milk of good quality—and by the administration of elixir ferri, quininæ et strychninæ phosphatum. In cases of scorbutic history, it is advisable to administer codliver oil, orange juice, lemon juice, and to give a mixed diet.

## GANGRENOUS STOMATITIS.

### (Noma; Cancrum Oris.)

This is a gangrenous inflammation of the cheek and adjacent parts of the mouth, occurring chiefly in young children living under extremely unhygienic conditions, or following the usual childhood diseases and intestinal disorders. It may be associated with syphilis, scurvy, typhoid fever, or small-pox. It is a rare disease, and is quite fatal.

Various pathogenic micro-organisms have been found in the tissues, among which are the Klebs-Löffler bacillus, diphtheroid organisms, and spirochetes. The disease begins in the mucous membrane of the cheek, near the corner of the mouth, as an ulcer, spreading rapidly to adjacent tissues, and a nodular sensitive growth may be felt between the palpating fingers. The skin of the cheek becomes reddened and brawny in color, and is greatly swollen. The entire thickness of the cheek then becomes black and gangrenous, often sloughing away so as to expose the interior of the mouth. These changes may take place in from twenty-four hours to three or four days. Shreds of tissue discharge from the gangrenous opening in the mouth, and there is a distinct foul odor in the diseased area. The gangrene may extend to the adjacent tissues, affecting the gums, the jaw, and the entire side of the face. It is not often that the opposite side is affected. The disease is attended with fever, ranging from 104° to 105° F. (40° to 40.4° C.), and the physical condition of the patient rapidly wanes. The disease is fatal in from 75 to 80 per cent. of the cases. If recovery takes place, it is accompanied by marked disfigurement.



### TREATMENT.

Every form of stomatitis occurring in children should receive immediate and constant medical attention. Hygienic conditions in the home demand careful attention, and the food should be carefully selected, and of wholesome, nourishing quality. Ulcers about the gums and cheeks are to be immediately cauterized with silver nitrate, and the mouth kept in a clean condition. Suspicion of gangrene calls for early and wide fulguration of the affected parts, advancing far into the healthy tissue. Cultures should be taken from the diseased parts. Diphtheria infection calls for the early administration of diphtheria antitoxin in doses of from 500 to 1000 units, and repeated as the individual case may require. If the disease has progressed, and extensive gangrene is present, the diseased area should be resected by the thermocautery and scalpel. The parts are dressed with a solution of potassium permanganate (1:500), or with the Carrel-Dakin solution. The mouth must be kept clean by spraying with liquor antisepticus alkalinus. General stimulants are indicated to build up the vitality of the patient.

### MERCURIAL STOMATITIS.

This occurs among workers in mercury (barometer makers, chemists, and others who directly handle this metal in the arts), and among persons under medication with this drug. The disease usually is associated with carious teeth, diseased gums, and foul, unhygienic condition of the mouth, and is found among those workers whose physical condition is below par. The gums are red, swollen, and tender, and there is increased salivation. There is a metallic taste in the mouth and the breath is fetid. Digestive disorders usually are associated, as well as physical and mental depression.

### TREATMENT.

The treatment calls for removal from the place of employment, or for the withdrawal of internal medication. Mercury must largely be eliminated through the bowels, so that saline cathartics are therefore indicated, such as Epsom salt, Glau-

ber's salt, and effervescing citrate of magnesia. Hot baths also may assist elimination through the skin. In cases of marked salivation, tincture of belladonna is indicated, 8 to 10 minims (0.50 to 0.60 mil), three times a day. A mouth-wash of potassium chlorate, 10 grains to the ounce (0.65 Gm. to 30 mls), or of the same strength of sodium bicarbonate may be of great value. In adults, potassium chlorate may be administered internally, 2 grains, every three hours. Persons working in industries where mercury is used should have their teeth constantly attended to, and should use an antiseptic mouth-wash as a routine preventive measure. Persons under mercurial medication should be cautioned about attention to the teeth and to the gums.

### CARRIERS.

Persons who harbor the germs of infectious disease in secretions of the nose and mouth or intestinal tract, and are not themselves affected by the presence of these micro-organisms, are termed carriers. The streptococcus, staphylococcus, diphtheria bacillus, pneumococcus, bacillus of Bordet and Gengou, influenza bacillus, tuberculosis bacillus, diplococcus intercellularis meningitidis, and the globoid bodies of infantile paralysis have been reported to exist in the secretions of the nose and throat of persons apparently in normal physical health. Such persons may innocently transmit these micro-organisms to others through the act of sneezing, coughing, spitting, or by the use of common drinking cups, glasses, eating utensils, and towels. When these bacteria are inhaled and find fertile soil for development in persons whose vital resistance is below par, clinical manifestations of their respective diseases develop. The carrier problem is a very difficult one to handle. Isolation of carriers from a public health point of view seems advisable and practicable in the presence of epidemics and endemics. It is difficult, and often impossible, to detect all carriers, inasmuch as it would take a numerous force of medical inspectors to survey and to examine a large proportion of the population when epidemic diseases prevail.

During the epidemic of poliomyelitis in New York City, Newark, and Philadelphia, in 1916, it was believed that the

carrier was largely responsible for the widespread distribution of this infection. During the epidemic of pneumonia and diseases of the respiratory tract in the northeast section of the United States, principally about New York and Philadelphia, during the winters of 1915, 1916, and 1917, it was also believed that the carrier was to be blamed for the great morbidity reported in these localities. Investigation among school children indicates that approximately 1 per cent. are carriers of diphtheria. This conclusion is based upon systematic inspection and culture of the throats of all children.

### TREATMENT.

The prevalence of pneumonia in the various training camps of the National Army has caused the Federal officials to make an intensive study of the carrier problem. It is believed that the streptococcus, which often is found normally in the secretions of healthy soldiers, may during the course of an attack of measles invade the pulmonary tissues and bring about a secondary pneumonia. The medical authorities are, therefore, seriously considering the prophylactic inoculation of every recruit with an antipneumococcus or an antistreptococcus serum in order thus to produce immunity against pulmonary infection with these micro-organisms.

It is essential that housing conditions of both the military and civil population be carefully supervised during an outbreak of epidemic disease, especially in the case of pneumonia. The barracks should be constructed so as to allow free ventilation and the constant circulation of fresh air. Where people must be quartered in large numbers, and where space is limited, constant ventilation is absolutely essential. Among the civil population the street cars should be thoroughly ventilated at all times, irrespective of the type of weather, and limited by the comfort and needs of the riding public. Educational propaganda along the lines of careless expectoration on the sidewalks and in public places is of great importance. The public should be encouraged to use the handkerchief during the act of coughing and sneezing, and should be taught the advantages of sleeping in well ventilated bedrooms.

Every health department should make efforts to control carriers by a system of inspection and quarantine. The best

results can be obtained in the case of typhoid and diphtheria carriers. In the case of the former, all food handlers should be licensed by the health department, and required to undergo a medical examination for the purpose of detecting typhoid in the stools. New York City has been very successful in detecting many carriers of disease in this way, and in preventing epidemics of typhoid by forbidding such persons to be employed or to carry on business in places where foods are handled or sold. In the case of diphtheria, all contacts should be isolated and the throats cultured, and all those giving positive results should be quarantined, and given such treatment as would be indicated in the general infections of the mouth.

The usual mouth-washes, as indicated in previous paragraphs, should be used for the treatment of carriers where the infection lies in the nose and throat. In the case of typhoid, however, persons should be instructed regarding the disinfection of the stool, careful cleansing of the hands, and to refrain from contact with foodstuffs intended for use by others. Internal medication to destroy the typhoid bacillus in such cases has not been very successful. During serious epidemics of diphtheria and cerebrospinal meningitis, carriers should be isolated until negative cultures are derived from the nose and throat.

### EPIDEMIC SORE THROAT.

Epidemics of sore throat have been reported from various communities, affecting both children and adults, and traced to infection of the milk from the udder of the cow. The streptococcus has been pointed out as the causative agent. The symptoms are those of a sore throat, enlarged tonsils, angina, high fever, and extreme prostration. Household epidemics of sore throat call for immediate investigation of the milk supply. A thorough and effective system of pasteurization would, of course, obviate any such spread of disease.

The *treatment* of the disease is the same as in any other infection of the throat. Local applications of 20 per cent. solution of argyrol, or 10 per cent. solution of silver nitrate, are recommended. Internally, a course of calomel followed by Epsom salts is effective in reducing the state of toxemia,

while a prescription calling for quinin 2 grains (Gm. 0.130), and strychnin  $\frac{1}{30}$  grain (Gm. 0.00216), three times a day, will directly combat the infection. A mouth-wash of liquor antisepticus alkalinus is also valuable. If the throat is very painful, the patient may be given pieces of ice to hold in the mouth, and drinks of orange juice or lemonade with shaved ice are very soothing.

### SYPHILITIC AFFECTIONS OF THE MOUTH.

The secondary stage of syphilis produces lesions of the mucous membrane of the mouth which are characterized by whitish or grayish patches on the lips, hard and soft palate, and tonsils, surrounded by inflammatory bases. Primary lesions may also occur in the mouth of the adult, most frequently on the lips, hard or soft palate, or on the tonsils. Nursing infants may also present primary lesions in any part of the mouth. The third stage or gumma formation is usually found on the hard or soft palate, the latter oftentimes being destroyed by degenerative processes, which result in regurgitation of fluids through the nose. The tongue also may become affected by gummatous degeneration with more or less hardening of the tissues.

### TREATMENT.

The treatment is a part of the general medication for the systemic infection of syphilis. Salvarsan, neosalvarsan, and the American products arseno-benzol and arsphenamin, licensed by the United States Government, are the preparations most valuable in the treatment, either in the secondary or tertiary stage. (See Vol. i, p. 80.) Iodids and mercury are indicated. Locally, the usual mouth-washes, such as Dobell's solution, liquor antisepticus alkalinus and equal parts of peroxid and listerine, may be used in the treatment of the mucous patches. Silver nitrate (10 per cent.) may also be applied daily to the ulcerated areas.

Patients should be instructed regarding the use of common eating utensils, handkerchiefs, towels, and drinking cups. In a number of States persons affected with active syphilis can-



not be employed in public eating places, restaurants, dining-cars, hotels, saloons, lunch counters, and other places where foods are sold. In many cities milk dealers and produce merchants are routinely inspected, and forbidden to carry on their business if they themselves are infected and come in contact with the merchandise. It is very important that persons in institutions, hospitals, and asylums, and in military camps be isolated when suffering from secondary lesions of syphilis. Extensive transmission of the disease has been known to occur where there has been laxity in these institutions in causing isolation of such patients. The so-called sanitary bubbling fountains have been condemned because active micro-organisms of syphilis have been found on the mouth pieces, in spite of the constant flushing with water. For the same reason common cigar-cutters have been condemned. The physician should in a sense act as a health officer and instruct his patients accordingly.

### GONORRHEA OF THE MOUTH.

Gonorrhea of the mouth may occur in infants from two to twelve days after birth. Infection in the parturient canal may be the exciting factor, although infection is known to occur through carelessness on the part of the nurse or mother in cleansing the mouth of the child. In adults the infection is characterized by marked inflammation and swelling of the soft parts.

Silver nitrate, 10 grains to the ounce (0.65 Gm. to 30 mils), should be applied to the ulcerated and inflamed surfaces twice daily. Glycerite of tannin (50 per cent.) may also be used for this purpose. The usual mouth-washes already recommended under this section are indicated, such as peroxid and liquor antisepticus alkalinus.

## DISEASES OF THE TONGUE.

### ACUTE GLOSSITIS.

Inflammation of the tongue, either acute or chronic, may exist alone or as a part of a general mouth infection. Acute glossitis affecting only the superficial structures of the tongue generally accompanies the acute stages of tonsillitis, stomatitis, pharyngitis, digestive disorders, and various systemic diseases, or it is directly the result of traumatism.

The tongue is slightly swollen, the epithelium is thickened and reddened, and the papillæ stand out prominently. There is a sense of thickness of the tongue, dryness, and sometimes pain on mastication or swallowing.

When the deeper structures of the tongue are affected, the condition may be attributed to marked traumatism from a bite or from irritation of a decayed or displaced tooth, or from burns of caustic drugs, poisons, and chemicals. Tuberculosis, syphilis, scarlet fever, typhoid fever, smallpox, and erysipelas may be attended with acute glossitis. The inflammatory process may be so great as to cause the tongue to protrude between the lips. The face seems full, and the general appearance of the patient may be changed. Deglutition and respiration may be very difficult; the temperature is elevated, and there is a feeling of weakness and discomfort. If the inflammatory process goes on to suppuration, the temperature and general symptoms are intensified. The submaxillary and sublingual glands become enlarged, and the chin appears double. The prognosis is usually favorable. When, however, the infection is rapid, the disease may extend to the glottis, with fatal consequences. If gangrene supervenes, the condition becomes intensely grave.

### TREATMENT.

In the mild catarrhal type, an alkaline mouth-wash used every two hours is beneficial. *Liquor antisepticus alkalinus* is as good as any. Sodium biborate and sodium chlorid, 5 grains each to the ounce (0.32 Gm. to 30 mils) of water, make an efficient lotion. The burning sensation of the tongue may be relieved by cracked ice.

When the deeper structures are implicated, the patient should be thoroughly purged with Epsom salts, citrate of magnesia, or gray powder, or by the use of compound cathartic pills. An ice-cap about the neck gives great relief. Cracked ice in the mouth is also beneficial. If the pain is severe, cocain (4 per cent. solution) may be painted on the tongue. For the swelling, adrenalin hydrochlorid applied to the surface of the tongue is recommended. If the process continues unabated, free incision on either side of the median line becomes necessary. An artificial leech applied to the side of the jaw may be used to relieve the congestion. Severe cases call for stimulation. The diet during the course of a severe infection should be liquid, and rapidly increased as the disease regresses. The cessation of symptoms is usually accompanied by desquamation of the epithelium of the tongue.

### CHRONIC GLOSSITIS.

Continued irritation of the tongue, caused by jagged teeth, alcohol, strong caustic drugs, the constant chewing of tobacco, or persistent gastro-intestinal disorders, tuberculosis and syphilis may result in a low-grade inflammation of the superficial or deep structures of the tongue. The epithelium is thickened; the heaping of the cells tends to obliterate the capillary interspaces, with more or less linear streaks of normal tongue surface, dividing it into irregular whitish areas. In other cases the epithelium is atrophied, with reddish patches, round, oval, or irregular, scattered over the surface of the tongue.

**Geographical Tongue.** This disorder is thus named because it appears to resemble geographical charts. There are various sized denuded epithelial areas, the centers of which appear to have normal epithelial cells. The outer borders are whitish or yellow, changing in appearance and spreading over the tongue. Desquamation may also occur on the edges of the tongue, causing it to appear roughened and red. This condition occurs chiefly in children, and is sometimes known as ringworm or eczema of the tongue. The subjective symptoms are a sense of burning and slight occasional pain.

In superficial glossitis and in geographical tongue the usual

alkaline washes already mentioned for mouth infections are recommended. A 2 per cent. solution of silver nitrate may be painted over painful or fissured areas. The exciting cause of the disease should be thoroughly investigated, more especially the teeth. If the tongue is *tuberculous* or *syphilitic*, as indicated by the history and laboratory findings, the treatment should be directed toward these special diseases.

**Gumma** of the tongue should be suspected if one or more localized round or flat areas of induration are associated with a definite history and laboratory findings of syphilis. These gummatous areas vary in color from pink to dark red; later they break down and ulcerate, leaving ugly, punched out, raw surfaces. Pain is slight or absent.

The internal administration of arsenobenzol or arsphenamine is indicated. The iodids are very valuable in gummatous formations.

**Tuberculosis** of the tongue manifests itself by the appearance of tubercles on the tip or edge of the tongue, with subsequent ulceration and the development of discharging painful denuded areas. These ulcerations should be treated by the local application of silver nitrate (10 per cent.), pure carbolic acid, trichlorascetic acid, or tincture of iodine.

**Leucoplakia** (white tongue) is a chronic superficial pathologic condition of the tongue manifested by the appearance of hard raised whitish patches, which are smooth or fissured. This condition is usually found among male adults who are habitual smokers, and in persons whose teeth are decayed or have irregular broken surfaces. Gastro-intestinal disorders, skin diseases, with special mention of psoriasis and other scaly conditions, may be associated. It is claimed that leucoplakia in a fair proportion of cases is followed by cancerous implantation. The condition begins as smooth red patches on the anterior surface of the tongue, and sometimes on the lips, cheeks, gums, and palate. These patches tend to coalesce, but usually are limited to the area of the tongue anterior to the circumvallate papillæ. They cause little or no subjective symptoms except when fissured, in which case there is more or less pain.

### TREATMENT.

The treatment should be directed toward eliminating the exciting causes. Sharp, irritating teeth should be removed, or properly attended to by the dentist. Smoking should be gradually reduced in frequency, and finally abandoned. The usual alkaline mouth-washes are valuable in that they keep the mouth clean, sweet, and fresh. Alcohol should be forbidden. Chromic acid solution (2 per cent.) may be applied locally over the areas to facilitate exfoliation of the plaques. Salicylic acid (1:1000) is also recommended for this purpose.

### SUBLINGUAL ULCER.

This condition is often found in children affected by whooping-cough. It is believed to be due to irritation caused by the teeth during the act of coughing. It is usually located at the frenum of the tongue. No special treatment is indicated other than the use of the usual alkaline mouth-washes.

### TUMORS OF THE TONGUE.

Carcinoma not infrequently affects the tongue. Pipe smokers and tobacco chewers are susceptible, and traumatism or constant irritation of the tongue predisposes to the stimulation of the tissues with cancer formation. Every ulceration or papillomatous growth of the tongue should be looked upon with suspicion as a precursor of a potential malignant growth. If associated with enlargement of the submaxillary or post-cervical glands, the diagnosis of cancer is very likely.

All ulcerations of the tongue should be treated with applications of silver nitrate, 10 grains to the ounce (0.65 Gm. to 30 mils), while all papillomatous growths of epithelial heapings should be examined microscopically by making sections of the affected parts. Cancerous processes should be treated either by fulguration or by excision of the tongue.

Among other growths of the tongue may be mentioned sarcoma, lymphangioma, hemangioma, lipoma, and fibroma. All these conditions are surgical and are therefore not to be considered here.



### MACROGLOSSIA.

This is a general enlargement of the tongue associated with cretinism. The treatment is directed toward the thyroid deficiencies. (See Vol. ii, p. 104.)

## DISEASES OF THE SALIVARY GLANDS.

### PTYALISM.

(Hypersecretion.)

An increased flow of saliva may occur during the menstrual periods, in the early days of pregnancy, in certain mental states, such as hysteria and mania, in exophthalmic goiter, and during the administration of drugs like mercury and iodids.

A moderate increase of saliva needs no special treatment, but when the condition become annoying and distressing to the patient, tincture of belladonna may be given, 5 to 10 drops (0.30 to 0.60 mil) every three hours, until comparative dryness is produced. It is necessary, however, to ascertain the cause in each instance, and remove it if practicable.

### XEROSTOMA.

(Dry Mouth.)

A decreased secretion of the salivary glands may occur after mumps, at the time of the menopause, in diabetes, and in neurasthenia. As a result of decreased salivation, the tongue becomes dry and fissured, and the entire mucous membrane becomes parched. The disease is rather uncommon. Salivation may be stimulated by the use of the faradic current over the salivary glands, and by the internal administration of pilocarpin nitrate,  $\frac{1}{20}$  grain (0.00324 Gm.) in water, three times a day.

## INFLAMMATION OF THE PAROTID GLANDS.

The subject of mumps has already been considered under the Specific Infections. (See Vol. i, p. 110.) The parotid glands may become the seat of inflammatory changes during the

course of the various infectious diseases, especially in typhoid fever and pneumonia. It may be secondary to suppurative conditions in the kidneys or liver, or follow certain abdominal operations. Infection occurs through the blood, or by continuity of tissue through Stenson's duct. The infection may result in enlargement of the gland, attended with pain on swallowing, with complete resolution in a few days. In other instances suppuration may occur, with rupture of the abscess mass, either externally through the skin, or internally into the mouth, ear, or even into the middle fossa of the skull. Infection may also travel through the tissues of the neck, extending as far as the clavicle, or even into the mediastinal spaces of the chest.

When parotitis occurs in the course of another infection, it causes an increased rise in the temperature, pain and discomfort on chewing, and prolongs the course of the original disease.

#### TREATMENT.

Great relief may be obtained from hot applications, either in the form of hot stupes (one tablespoonful (15 Gm.) of Epsom Salts to a quart (1 l.) of hot water), hot water bottle, electric pad, flaxseed poultice, or hot salt bag (salt heated in pan and then placed in original salt bag). Ichthyol ointment may also be applied. When the affection has progressed to the stage of suppuration, the pus must be evacuated by an incision over the most prominent part of the fluctuating mass. While free and early incision is recommended in ordinary abscess of other parts of the body, it is advisable in this instance that incision be delayed until the abscess mass points, in order to avoid a sinus formation, which may result when cutting into healthy glandular tissue. In the case of females, it is often advisable to open the abscess mass through the mouth to avoid the possibility of scarring the face.

#### SALIVARY CALCULI.

Salivary calculi, composed of calcium carbonate phosphate, may occasionally be found in the salivary ducts or in the substance of the glands. The treatment is surgical.

### LUDWIG'S ANGINA.

Ludwig's angina is a cellulitis of the floor of the mouth, characterized by swelling of the submaxillary regions, either unilateral or bilateral, which later may extend to the soft tissues between the mandibles, and reach the anterior surface of the neck as far as the sternum. It is an infection of the submaxillary glands and adjacent cellular tissue, caused by the invasion of pathogenic micro-organisms through the lymphatics. An abrasion in any part of the mucous membrane of the mouth or a carious tooth may be the precipitating agent.

At the onset of the infection the fever is moderate, but rises as the disease extends, especially when attended with abscess formation. The skin surface of the chin becomes tense and full, edematous, and, later, hard to the touch. Chewing and swallowing become painful. If the disease extends, as it may, to the larynx, or to the lungs, symptoms of asphyxiation become prominent.

*Treatment.* Hot applications are quite acceptable. Stupes of Epsom salts applied every twenty minutes in the hour tend to favor resolution. Hot and cold applications may be alternated. When the swelling is intense, with or without suppuration, deep incisions on either side of the median line should be made to drain the parts of their infectious material.

## DISEASES OF THE ESOPHAGUS.

### ACUTE ESOPHAGITIS.

Being the passage-way of food and drink, the esophagus may be subject to traumatism and injury by irritating drinks, caustic liquids, sharp-edged solid foods, foreign bodies, and the extension of inflammation from adjacent parts. Acute infection may result during the course of acute systemic diseases, such as syphilis and tuberculosis. Diphtheria and thrush may also inflame this region in rare instances. Injury to the gullet by foreign bodies is quite common in children. In adults, injury occurs among dressmakers, who constantly hold pins in their mouths; among window-dressers, carpet or

oilcloth layers, who have the habit of holding tacks in the mouth; and among the insane, who may swallow various objects, varying in size from tacks and pins to large nails, screws, and other metal objects.

The characteristic symptom is pain, exaggerated on taking food. In severe types all food may be rejected by retching and vomiting.

#### TREATMENT.

The treatment, of course, calls for the removal of the cause, followed by careful dietary of non-irritating substances. At first lukewarm foods should be given, such as milk, broths, beef tea, weak tea, and later there may be added buttermilk, gelatin, junket, custards, and tapioca pudding. If the pain is severe, all food should be withdrawn for from twenty-four to forty-eight hours. Enteroclysis of salt solution may be substituted to supply the necessary fluid for the body. To allay the acute pain, olive oil, cream, or milk may be used as suitable demulcents. Intense burning of the esophagus is usually associated with some condition of the mouth, which makes swallowing very painful, and oftentimes very difficult. In such cases bismuth subcarbonate 20 grains to the ounce (1.3 Gm. to 30 mils) of water may be administered slowly as a protective coating. Sippy<sup>8</sup> recommends the administration of a teaspoonful (3.7 mils) of adrenalin hydrochlorid, 1:1000, containing 1 per cent. cocain, just before feeding.

Cicatrization of the tissues following caustic burns is inevitable. It is, therefore, advisable to use esophageal bougies about ten days after the injury to prevent the possibility of stenosis. The size of these dilators should be judged from the extent of the scar tissue contraction, and gradually increased to the full diameter of the esophagus.

#### ULCER OF THE ESOPHAGUS.

Erosions of the mucosa and subjacent tissues of the esophagus may follow simple esophagitis, or may result from the corrosive action of poisons and acids, from the abrasion of foreign bodies, from the invasion of disease processes such as syphilis, tuberculosis, or actinomycosis, from the pressure of growths in neighboring organs, from regurgitation of di-

gestive juices of the stomach, and from a lowered vital state during the course of acute affections or chronic cachectic states. Pain is a prominent symptom, and occurs immediately after deglutition. Localized behind the sternum, and referred to the back, it may be difficult to take solid foods, or even liquids. The passage of food over the irritated surface may cause spasm of the esophagus, resulting in regurgitation of food. The vomitus may be blood-stained, or may contain bright red blood. The ulcerated area on some occasions ruptures, and this is attended with hemorrhage into the stomach or externally through the mouth. In such an event the prognosis is less favorable.

Ulceration of the esophagus at the lower third may occur as the result of insufficiency of the cardiac sphincter, permitting the gastric juices to enter the esophagus. The lesion in this area is called peptic ulcer, and is infrequent in occurrence. In this instance the pain is most acute at the end of deglutition, and is localized over the end of the sternum, and referred to a corresponding area over the spine.

Tuberculous and syphilitic ulcers are rare, and are diagnosed by appropriate laboratory tests.

#### TREATMENT.

In cases of mild ulceration, liquids foods are tolerated. Severe cases, however, may require the withdrawal of all foods by mouth for from twenty-four to forty-eight hours. In this instance fluid may be supplied to the body by enteroclysis, and occasional rectal feeding with peptonized milk. When the pain subsides, the patient may be given milk, junket, gelatin, tapioca pudding, soup, meat broths, ice cream, and water ices. Bismuth subnitrate or carbonate, 10 to 20 grains (Gms. 0.65 to 1.3), may be given three times a day in water or milk. Rest in bed is quite essential during the acute inflammatory stage. Ulcerations at the cardiac end of the esophagus are to be treated in like manner as those occurring in the cardiac end of the stomach. (See Gastric Ulcer, Vol. ii. p. 695.) If the disease is of such an extent that the patient is unable to take nourishment for an extended period of time, and is attended with loss of weight, and in cases where hemorrhage is severe, it is advisable to perform a gastrostomy.



## CARCINOMA OF THE ESOPHAGUS.

The most frequent, serious, and fatal disease of the esophagus is cancer. It occurs most frequently in males over forty years of age. The lower end of the esophagus is most often affected, although a large number of cases occur in the region of the bifurcation of the trachea, and a smaller number in the cervical region. Their structure may be of the soft medullary type, or fibrous in character. The growth usually encircles the esophagus, causing varying degrees of stenosis. The pathology depends upon the type of the growth and the predominant structures. It does not differ materially from cancer in other parts of the body. Carcinoma selects the esophagus because this organ is frequently subject to traumatism, and because its embryonic structure may include a few superfluous cells which later develop into a tumor mass of the entoderm. The most prominent symptom is dysphagia, which becomes more marked as the stenosis increases. Vomiting of mucus, blood, and disintegrated tissue occurs. The cervical glands become enlarged. Metastasis takes place in the liver, lungs, pleura, pericardium, and the lymph nodes along the thoracic aorta. The disease is generally fatal in from six months to one year.

Ulceration and maceration of the tumor mass may be attended by perforation into a bronchus, the aorta, or pericardium, causing extensive hemorrhage and bloody vomitus. As the disease progresses, feeding becomes more difficult. There is extreme loss of weight from inanition and cachexia, and the patient presents symptoms as in carcinoma of other regions. This disease oftentimes simulates simple stricture, caused by pressure from adjacent organs, or by mechanical or chemical irritation. The diagnosis, however, is determined by the progressive cachexia in a patient past middle life, and the finding of carcinoma tissue in the vomitus. X-ray diagnosis is of great importance in the differentiation.

### TREATMENT.

Early diagnosis calls for careful and judicious manipulation by bougies. This may give material comfort to the patient by stretching the contracted lumen. Since maceration

of the diseased tissues may be caused by passing bougies, it is advisable that their use be restricted to those familiar with the procedure. Dilatation should be performed weekly. In spite of these treatments, however, which are only palliative, the dysphagia increases, and the vomiting and bleeding become more frequent. Every possible measure should be adopted to give the patient comfort and ease. The food should be liquid and nourishing, consisting of articles such as milk, buttermilk, eggs, custards, gelatin, tapioca pudding, floating island, ice cream, and water ice. Proper and constant nursing tends to make the patient more at ease.

If there is considerable pain, rectal feeding with peptonized milk should be given for several days. If the stricture has so far progressed as to cause rejection of all food, and emaciation increases, a gastrostomy is demanded. The surgical treatment of this condition has not as yet developed sufficiently to risk the opening of the chest and resecting the diseased portions of the esophagus. If there is severe hemorrhage, and starvation is threatened, surgical intervention is indicated, with a view to creating an artificial sinus between the stomach and abdominal wall, through which the patient may be fed. In some instances the esophagus immediately above the stricture becomes dilated, creating a pocket where food lodges and becomes decomposed. Mucus, pus, and blood may also accumulate in this dilated portion. In such instances the esophagus should be washed out once daily with salt solution or boric acid. The *x*-ray treatment may be of some value in the hands of experts.

Much has been done recently along the lines of radium therapy. This preparation, however, is quite expensive, and is not within the reach of all. Its use is restricted to those who are most expert in handling it.

### ESOPHAGISMUS.

Occasionally the muscular structure of the esophagus is subject to tonic or clonic contraction, which is precipitated reflexively by ulcers of the larynx, gastric disturbances, uterine or other abdominal irritations. It may be an accompanying symptom in hysteria, neurasthenia, tetanus, hydrophobia, epi-

lepsy, and highly neurotic states. The principal symptom is dysphagia. While liquids can generally be taken, solids cannot. There is pain over the sternum, and a sense of choking. Other symptoms are those referable to the cause producing the esophagismus. Spasm may occur at the pharyngeal or cardiac end, or at any place in its length, the first two sites being the most common.

Obstruction of the pharyngeal end is readily overcome by the passage of large-sized objects. When it occurs in the course of the esophagus, the systematic passage of bougies readily overcomes the difficulty. Sometimes medication with bromids will be sufficient to cause relaxation. The systemic condition of the patient should be treated as a preventive measure from future attacks. Stricture of the lower end is commonly called cardiospasm, and is discussed under Diseases of the Stomach (*q.v.*).

### DIVERTICULUM OF THE ESOPHAGUS.

Sacculation of any part of the esophagus may occur as the result of pressure or traction upon its walls. The pouchings are anatomically constructed of mucous membrane and connective tissue. It is in reality a hernia, which pushes aside the muscle fibers retaining its coat of mucous membrane and submucous tissue. The contents of the sac may be either fluid, solid, or both. A congenital deficiency of the brachial clefts of the embryonic structures may predispose to sacculation. Acquired hernias of the esophagus are due to pressure from within, with lowered resistance of the muscular tissue. Repeated pressure of solid foods over the same weakened area constantly increases the sacculation, until a well-formed, pear-shaped diverticulum results. Injury or previous inflammation may be a contributing cause. Sacculation occurs most frequently at the junction of the pharynx and esophagus in the posterior aspect, but may also occur in the pharynx near the bifurcation of the trachea, and above or below the left bronchus. It occurs more frequently among males of middle adult life.

Traction diverticula result from adhesions of adjacent tissues to the esophagus following inflammatory lesions, such

as bronchial adenitis with suppuration. These diverticula are funnel-shaped, and are located in the upper end of the esophagus, anterior and near the tracheal bifurcation. They do not usually produce subjective symptoms, while the pressure hernias (less common in occurrence) give rise to well-defined clinical symptoms. If ulceration takes place in traction sacculations as the result of retained food particles, perforation may take place into the bronchi, pleural sacs, or rarely into the pericardium.

Sacculations resulting from pressure of food may often escape notice if very small, but give rise to a chain of clinical symptoms when large. Painful swallowing is commonly complained of, but its degree depends upon the size of the sacculaton and its location. As the diverticulum increases in size, vomiting becomes more frequent, with symptoms of threatened strangulation. The vomited material contains no gastric juice. This special point tends to differentiate gastric disorders. When the sacculaton becomes especially large, a pear-shaped mass may be palpated in the side of the neck. Pressure on the larynx causes dyspnea, pain, spasmodic coughing, and changes in the voice. The patient himself may complain of food lodging in a certain part of the esophagus. The condition tends to progress, the symptoms become exaggerated, and malnutrition results.

The diagnosis is made by the passage of a sound, which is arrested by the sac. Very often the sac orifice is small or tortuous, in which case the investigating bougie may enter the stomach directly. After eating, the sac usually enlarges and may be emptied by pressure of the hand. A bismuth meal followed by x-ray and fluoroscopic examination may locate the lesion.

Unless the condition is relieved by surgical means, death takes place by starvation or intercurrent affections. The patient, however, should be instructed to eat liquids or semi-solids, and to do so very slowly, in small quantities, and at frequent intervals. Retained and decomposed food in the sacculaton should be washed out daily by irrigations of boric acid solution or potassium permanganate (1:1000). When located in accessible regions, surgical intervention may be attempted. When the sacculaton increases so as to impinge

upon the trachea, the esophagus, and adjacent organs, the prognosis becomes very grave. The lumen of the sac may become obstructed by twisting or by pressure, in which case it becomes difficult to remove the contents. The patient should try feeding in various postures to encourage peristalsis of the esophagus. It may be necessary to pass a perforated bougie for the purpose of administering nourishment. If starvation is threatened, gastrostomy must be performed.

### FOREIGN BODIES.

Foreign bodies are frequently swallowed by children, and may become lodged in any part of the esophagus, most frequently below the cricoid cartilage. Pennies, buttons, bones, stones, pins, and various other objects have been found lodged in the esophagus. Adults may accidentally swallow safety-pins, false teeth, fish bones, peach stones, and other objects. If not removed early, they may cause damage to the esophageal walls, and produce ulceration of the tissues, edema, necrosis, and infection of the adjacent tissues. Death may result from extensive edema, obstruction, and infection. For this reason it is absolutely essential that all foreign bodies should be removed as soon as possible after the accident.

Pennies and safety-pins frequently lodge below the cricoid cartilage, and require great skill on the part of the operator to remove them. An especially devised esophageal forceps is required to extract foreign bodies. The esophagoscope may be employed to explore the esophagus and to direct the forceps to grasp the foreign body. The *x*-ray is a valuable aid in locating the foreign bodies capable of casting a shadow. The fluoroscope is also invaluable to note the progress made during the intervals of attempted extraction.

### STRICTURE OF THE ESOPHAGUS.

The lumen of the esophagus may be constricted at any part of its length from the pharynx to the stomach. The causes may be divided as those from within and those from without. Strictures from within may be the result of:

1. The corrosive action of acids.
2. Cicatrization of ulcers.



3. Obstruction by foreign bodies.
4. Cancer.
5. Syphilis and tuberculosis.
6. Polyps and diverticula.
7. Spasm of the esophagus.

Among the causes from without may be mentioned the various growths or malpositions of viscera:

1. Aneurysm.
2. Mediastinal tumors.
3. Thyroid and thymus tumors.
4. Enlarged cervical glands.
5. Fracture of the sternum, ribs, or posterior displacement of the sternum end of the clavicle.
6. Vertebral abscess.
7. Displacement of the heart by pericardial exudates.
8. Cicatrices in the tissues of the esophagus.

It is often very difficult to determine the nature of the obstruction. A careful history, however, may aid in the diagnosis. Slight stenosis may cause only a mild discomfort during swallowing, giving rise to a sense of pressure of obstruction behind the sternum, especially after eating solid foods. As the stenosis progresses there is a sense of obstruction in the epigastrium. The patient often notices that it takes a long time for the food to reach the stomach, and that the meal period is prolonged. Regurgitation of the food may occur immediately after eating, or several hours later. Pain is present in ulcerative or inflammatory stenosis, while in other instances pain may be absent. As the stenosis increases, the symptoms become exaggerated, and are accompanied by malnutrition and debility. The diagnosis may be confirmed by watching the peculiar attitude of the patient on swallowing. The passage of an ordinary stomach-tube may be unhindered in cases in which the stenosis is very slight. Moderate constriction, however, may be detected by the passage of a medium-sized olive-tipped esophageal bougie, the precise location of the obstruction being marked on the bougie when the tip becomes arrested. The degree of stenosis is determined by the passage of sufficient and various sized bougies until complete passage is facilitated. These manipulations, however, should only be performed after excluding aneurysm as a

cause of the obstruction. On auscultation over the left side of the spine a splashing or a flowing sound may be elicited at the site of the obstruction when the patient drinks water. Differentiation of the various forms of stricture must be made by the history. Esophagismus is most frequently found in neurotic females, and may be relieved by the systematic passage of bougies. Gummatous and tuberculous conditions give their respective history and physical findings. Corrosive strictures usually give other evidence of burns about the mouth. Malignant obstructions are most frequent in persons over the age of forty, and are accompanied by progressive signs of cachexia.

The prognosis of cancerous stricture is unfavorable. While death may be deferred by our advanced methods of treatment, permanent cure is out of the question. Simple strictures may give favorable results after a systematic course of dilatation, but most strictures tend to end fatally.

Cicatricial stenosis are next in frequency to those caused by cancer. Under routine and regulated dilatation, fairly satisfactory results may be obtained. Dilating bougies should be used as early as the first week following caustic burns. After the acute symptoms have subsided, an olive-tipped bougie should be passed twice weekly, increasing the size of the bulb until the full-sized tip makes easy passage. It may be necessary to continue the dilatation every month for one or more years, as the individual case may require. In cases of spiral or irregularly shaped strictures, which occur not only in the lumen but in the length of the esophagus, it may be necessary to resort to the threaded olive bulb, as advocated by Sippy.

**Method of Passing Whalebone or Steel Rod Bougie.** The patient should be given a half-ounce (15 mils) of olive oil before the operation, in order to facilitate the passage of the bougie. A 4 per cent. solution of cocain may be sprayed over the pharynx to prevent spasm and gagging. Place the patient in a comfortable low seat, the head thrown slightly backward, and held by an assistant, who faces the patient. The operator, standing beside the patient's head, inserts the finger of the left hand into the mouth so as to depress the tongue until the epiglottis is clearly seen. With the right hand the operator inserts the olive-tipped bougie along the epiglottis, direct-

ing it into the esophagus. Care should be exercised not to make a false passage into the larynx. The bougie is passed with steady and light pressure. The touch of the operator will indicate when the bulb is grasped by the esophageal muscles and directed downward. Just below the cricoid cartilage the tip is engaged tightly by the muscles. If an obstruction is met, the passage is hindered, or it may be firmly gripped or entirely obstructed. Mark the bougie even with the teeth, in order thus to locate the distance of the obstruction. The normal anatomic locations are as follows: From the teeth to the cricoid cartilage, 7 inches (17.7 cm.); to the left bronchus, 11 inches (27.8 cm.); and to the diaphragmatic opening, 15 inches (37.9 cm.).

## DISEASES OF THE STOMACH AND DUODENUM.

### ULCER OF THE STOMACH AND DUODENUM.

The term gastric or duodenal ulcer is applied to a solution of continuity of the mucous membrane of the stomach or the duodenum, with a tendency toward a penetrating disintegration and necrosis of tissue. Clinically, there is a striking predilection toward sudden remissions of symptoms with equally sudden exacerbations.

Ulcers may vary from the superficial erosions, described by Dieulafoy, through the phagedenic variety, commonly seen in chlorotic young women, to the true peptic ulcer, more common in men, which has a tendency to a deeper penetration and greater chronicity, with the formation of indurated scar-tissue edges.

Three *etiologic factors* have been generally understood to contribute most largely to this condition: (1) Failure of proper nutrition at a localized point of the mucous membrane. (2) Diminished resistance of this circumscribed area to the action of the peptic power of the gastric secretions, whether normal or increased. (3) The effect of the mechanical activity of the stomach musculature in the pyloric and prepyloric portion, where the greater majority of these ulcers occur. More recent experimental work has shown that there are two

further etiologic factors of importance that must be considered: (1) a toxemia of either metabolic or bacterial origin, and (2) the contributing effect of an increased intragastric tension, excited by spasmodic closure of the orifices of the stomach.

Turk has experimentally produced gastric ulcer by the intravenous injection of the colon bacillus. More recently Rosenow has reported that he could produce gastric and duodenal ulcer with considerable regularity by the intravenous inoculation of certain strains of streptococci, and he lays emphasis on the tonsils as being an important source of infection. Nevertheless, it would appear that the human gastric mucosa is much more resistant to true bacterial infection than is that of the experimental animal; nor is it likely that different strains of the same bacterial group, so beautifully shown by Rosenow in his experimental studies to have definite selective affinities toward the production of gastric and duodenal ulcer and appendicitis, can so uniformly produce similar conditions in the human being. Approaching the problem from the standpoint of a metabolic toxemia, Gundermann has experimentally produced both acute and chronic ulcers of the stomach by ligation of the left hepatic branch of the portal vein, and from his experiments he concludes that hepatic toxemia can initiate the ulceration. These experimental studies strengthen the suspicion that the close association existing between the simultaneous occurrence of a gastric or duodenal ulcer with a chronically inflamed or obliterated appendix, and somewhat less frequently with gall-bladder disease, is something more than mere coincidence.

Circumscribed malnutrition of the gastric or duodenal mucosa may be caused by localized circulatory failure, as a thrombosis or embolism of the terminal arterioles, or by sudden increase in intragastric tension, whereby the blood-supply is diminished or cut off; by the action of corrosive substances, and by the selective action of toxins from severe burns. Or the malnutrition may arise from extragastric organic disease, giving rise to passive congestion of the gastric capillaries, with the formation of petechiæ, submucous hemorrhages and infarcts; and, less commonly, from traumatic influences, chemical, biochemical or parasitic, from within or from

without, as from sudden blows or long-sustained pressure over the stomach, such as is seen in tailors, cobblers or weavers.

It can no longer be maintained that hyperchlorhydria is an essential in the production of the ulcer, although it doubtless does interfere with the ready healing of eroded spots, and in some cases may predispose to its causation. While hyperacidity is the rule and speaks in favor of ulcer, yet it is well to remember that many cases of gastric and duodenal ulcer occur in the presence of normal acidity, some with subacidity, but few, if any, with anacidity.

While it is a common disease, it is difficult to accurately estimate its *frequency*. Even in recent compilations the surgeon's statistics differ widely from those of the clinician, and it is not difficult to explain this. The surgeon's statistics show a lesser frequency, inasmuch as they deal only with those cases that come to the operating table, whereas the clinician sees many more cases, particularly those of the acute type which respond to medical management. Of course, there may be, and doubtless are, a certain number of errors in the clinical diagnoses, but this applies surgically as well as medically, for in these days there is an increasing number of clinically proven cases which fail to be corroborated by the exploring eye or finger of the surgeon, which later on develop unmistakable clinical symptoms, such as hemorrhage. In many such cases the failure of surgical corroboration is due to *insufficient exploration*, especially in ulcers located on the posterior wall, or to the inability to detect minute ulcers capable of giving rise to symptoms, but not sufficiently progressed to give evidence of scar-tissue contracture when viewed from the serosal surface only. The tendency of some of our master-surgeons to do an intragastric or intraduodenal exploration where the clinical evidence is sound is a tendency to be commended, provided that if in the future such a procedure can be shown not materially to increase the mortality. The true frequency of gastric and duodenal ulcer can best be gaged by the finding at autopsy of gastric or duodenal ulcers in the healed, quiescent, or active stages. From the autopsy records of many thousands of cases, Welch and Fenwick respectively estimate its frequency as being 5 per cent. and



4 per cent., although it could be determined as the actual cause of death in less than 1 per cent.

Probably 80 per cent. to 85 per cent. of chronic ulcers are found on the lesser curvature, near the pylorus. They are likely to straddle the lesser curvature, and usually show more extensive implication of the posterior than the anterior surface. From the scars and open ulcers found at autopsy, it is probable that gastric ulcer is much more common than duodenal, and that it has a much greater tendency to spontaneous cure, which somewhat explains the surgical statistics which show that duodenal ulcers are twice as common as gastric ulcers. According to Mayo's statistics, the site of the duodenal ulcer, in 96 per cent. of their cases, was in the first portion of the duodenum within 1 inch of the pylorus.

The size may vary from scarcely visible erosions to ulcers measuring 3 to 5 centimeters (1.18 to 1.96 in.) in diameter, with occasional reported cases of ulcers double this size. Chronic ulcers are more commonly large than are the acute ones. Duodenal ulcers are usually small.

Duodenal ulcers are almost always solitary, whereas in 20 per cent. to 25 per cent. of gastric-ulcer cases multiple ulcers are found. In about 3 per cent. of the Mayo's cases both gastric and duodenal ulcers were found, the former probably being primary.

As to sex incidence, statistics vary. Lockwood summarizes it as follows: "Acute ulcer of the stomach is three times as common in women as in men, that in chronic ulcers of the stomach the proportion between the two sexes is equal, while in chronic ulcer of the duodenum three-fourths of the cases occur in men."

Gastric and duodenal ulcers are common in early adult life, most acute ulcers occurring in the second or third decade, and most chronic ulcers giving rise to symptoms in the third and fourth decade. Duodenal ulcers may occur in the very young, Collins having reported that in 279 cases collected by him 15 per cent. were under 10 years of age, and Moynihan has called attention to the frequency with which duodenal ulcer may be the cause of *melena neonatorum*.

The earlier an ulcer is recognized and placed under proper treatment, the more likely is it to heal rapidly, and to stay

healed. Even in the absence of all treatment, or in the face of poor treatment, many ulcers will spontaneously heal, or, at any rate, fail to give rise to further symptoms. The more superficial the ulcer and the better its blood-supply, the more rapidly will it heal, with or without treatment; the deeper the penetration of the ulcer, the more indurated its edges, and the extent of its devascularity, the more resistant it becomes to medical measures. As to duration, ulcers may run a course of from a few weeks to many years. Histories of twenty years standing are not infrequent, and some cases may have lasted even longer. It is a disease, perhaps, most signally characterized by its chronicity when untreated, and by the exacerbations and remissions in its clinical symptoms.

The *complications* incident to any given case serve to modify the prognosis materially. The most important complications are pyloric obstruction from cicatrix, inflammatory edema or pylorospasm; continued hypersecretion, perigastritis or perigastric adhesions, perforation and carcinomatous degeneration. The last mentioned is the most dangerous, and, therefore, the most important. According to Wilson and MacCarty,<sup>9</sup> it is the most frequent complication of gastric ulcer, occurring in from 40 to 50 per cent. of cases, but such degeneration is rarely seen in duodenal ulcer.

Criticism of these statistics has arisen on the grounds that this percentage of incidence of carcinomatous degeneration is much too high. From the clinician's standpoint this is beside the point. Even should the true incidence be nearer 25 per cent. than 50 per cent., it is sufficiently high to make us keenly alive to its seriousness, and if the time now spent in splitting hairs over such a statistical controversy were applied to a closer study of our patients, and our energies bent to an elaboration of better *early* diagnostic tests, it would result in far more profit to both patient and physician.

As to the *mortality*, comparative statistics are unreliable, for the available data vary within wide limits in the hands of experienced observers and compilers. The surgical mortality depends primarily upon the surgical experience and capability of the individual operator, and secondarily upon the type of surgical complications, the duration of their existence, and upon whether or not the cases for operation are

selected. In the hands of a master-surgeon the mortality is extremely low. The writer is permitted to publish the following statistical tables of the total number of cases of gastric and duodenal ulcer operated upon by John B. Deaver during the past six years, 1909 to 1915, inclusive. During this time he has operated upon 43 gastric ulcers (unperforated) with 2 deaths, or a mortality of 4.6 per cent.; 179 duodenal ulcers (unperforated) with 6 deaths, or a mortality of 3.35 per cent.; 9 gastric ulcers (perforated) with no deaths; and 34 duodenal ulcers (perforated) with 2 deaths, or a mortality of 5.9 per cent.; and taking the total number of perforated ulcers, gastric and duodenal, with 2 deaths, the mortality is reduced to 4.6 per cent., and, as will be seen by consulting Table No. V, the mortality occurred in cases in which perforation had taken place more than thirty hours prior to operation. The complete tables are as follows:

LANKENAU HOSPITAL STATISTICS.

January, 1909—July, 1916.

TABLE No. I.

GASTRIC ULCERS.

Operation.	Number.	Recovered.	Died.	Mortality.
Post. gastro-enterostomy .....	21	21	0	0
Partial gastrectomy .....	13	12	1	7.7 %
Pylorectomy .....	2	2	0	0
Excision .....	5	4	1	20 %
Circular resection .....	2	2	0	0
Total .....	43	41	2	4.6 %

TABLE No. II.

DUODENAL ULCERS.

Operation.	Number.	Recovered.	Died.	Mortality.
Post. gastro-enterostomy .....	147	144	3	2 %
Partial gastrectomy .....	5	5	0	0
Pylorectomy .....	24	22	2	8.3 %
Excision .....	3	2	1	33⅓ %
Duodenorrhaphy .....	0	0	0	0
Total .....	179	173	6	3.35%

TABLE No. III.

## PERFORATED GASTRIC ULCER.

Operation.	Number.	Recovered.	Died.	Mortality.
Post. gastro-enterostomy .....	6	6	0	0
Gastrorrhaphy .....	3	3	0	0
Total .....	9	9	0	0 %

TABLE No. IV.

## PERFORATED DUODENAL ULCER.

Operation.	Number.	Recovered.	Died.	Mortality.
Post. gastro-enterostomy .....	24	23	1	5.1 %
Partial gastrectomy .....	4	4	0	0
Pylorotomy .....	1	1	0	0
Duodenorrhaphy .....	5	4	1	20 %
Total .....	34	32	2	5.9 %

TABLE No. V.

## PERFORATED DUODENAL AND GASTRIC ULCERS.

Time elapsing between perforation and operation:

## DUODENAL ULCER.

Hours.	No. of Cases.	Deaths.
1— 2	1	0
3— 6	9	0
7—12	8	0
13—24	7	0
2— 3 days	4	1
More than 4 days	5	1

## GASTRIC ULCER.

3— 6 hours	3	0
7—12 hours	4	0
13—24 hours	2	0

Total	43	2 = 4.6%
-------	----	----------

*End Results.* Traced 13 cases (11 duodenal—2 gastric), all well without return of symptoms.

No inquiry was sent to 3 cases operated in 1916.

It will be seen from a review of these statistics that the mortality is exceedingly low, and illustrates what can be accomplished by a master-surgeon of long experience. These statistics, however, cannot be taken as representative of the

surgery throughout the country, which, while steadily improving, has not reached a point that can compare statistically with the foregoing.

This is a reiteration of what the writer has frequently emphasized, namely, that in the selection of a surgeon for operations of the stomach or upper intestinal tract, other things being equal, a man should be chosen whose formative period of perfecting his surgical technic has been passed, and whose judgment is a result of years of surgical experience.

As a fundamental antecedent to correct *treatment*, correct diagnosis is indispensable. Differential diagnosis is often exceedingly difficult. The symptoms commonly ascribed to gastric and duodenal ulcer are very frequently simulated by many extragastric organic lesions, which reflexly give rise to functional disturbances of the stomach. There should be a close co-operation, in all such cases, between the clinician, the surgeon, the laboratory expert, and the röntgenographer, for many of these cases need intelligent, systematic investigation. The custom, so prevalent in the past, to use the exploratory laparotomy, as a means of making the diagnosis, is a measure to be strongly deprecated, and happily surgical interference of this sort is becoming less frequent. Once the diagnosis has been soundly established, the next important point is to determine whether the case in hand should be treated medically or surgically, and, if the latter, whether a laparotomy should be done at once, or whether a preliminary medical plan should be adopted for a trial of several weeks or several months. To settle this the clinician must establish the following points: How long has this ulcer existed? Is it an open or a closed ulcer? A bleeding or non-bleeding one? And are there complications?

Among the complications and sequelæ the following must be considered:

1. Carcinomatous degeneration of a gastric ulcer. This is the first and most important complication, and a frequent one, when the ulcer is situated on the gastric side of the pylorus, and, aside from perforation, is the one immediate indication for prompt surgical intervention. Where the ulcer is on the duodenal side of the pyloric ring, the chance of carcinomatous degeneration is comparatively remote.



2. Pyloric obstruction. Here one should differentiate obstruction caused by (a) a stenosing cicatrix shutting off the gastric or duodenal lumen; (b) inflammatory edematous swelling; (c) perigastritis, with or without adhesions; (d) pylorospasm.

3. Hypersecretion.

4. Perigastric abscess, with or without a localized peritonitis.

5. Various contracting deformities, other than pyloric, such as hour-glass contraction.

6. Hemorrhage: acute or by continued minute oozing.

To determine the various complications may tax one's diagnostic acumen to the utmost, but upon it depends largely the degree of success in the treatment of such cases.

A word here might properly be said in regard to the proper *diagnostic maneuvers*. A careful examination should be made of both gastric and duodenal sediments, for blood, pus, turbid bile, bacteria, epithelial desquamation, etc.<sup>10</sup> One or more string tests to aid in the localization of the ulcer and to determine whether it is bleeding; extraction of the gastric contents to determine hypersecretion should be made at the following hours of the fasting stomach: 6 to 7 A.M., 11 to 12 A.M., 5 to 6 P.M., and 11 to 12 P.M. These extractions may best be made with any small-bore tube, with a proper metal tip, and aspirated by means of a syringe; careful examinations, chemical, bacterial and cytologic, and, finally, to support and corroborate these facts by means of a properly made *x-ray* examination of the intestinal tract, in the hands of an experienced actinologist.

The Gluzinski method of testing the gastric secretory response to different test-meals is one of considerable aid in differentiating uncomplicated gastric ulcer from one undergoing carcinomatous degeneration.

The cholesterol content of the blood should be estimated as a differential test between cholelithiasis or cholecystitis with adhesions to the stomach or duodenum, simulating obstructing duodenal ulcers; likewise, in doubtful cases, an estimation of the efficiency of the pancreatic ferments should be made, both in the aspirated duodenal contents and in the stools; and in all cases in which a syphilitic history is

obtained a Wassermann reaction should be carried out, and, if positive, energetic antiluetic measures should be begun and continued for a reasonable length of time before operative procedures are considered. Naturally, all of these special tests should be preceded by a careful anamnesis, and a complete physical examination, including blood-counts and urinalyses.

In cases that are diagnostically so clear, such as those with a history of a profuse hematemesis (where esophageal varices can be excluded), or a massive melanic stool, one may not have to resort to these finer differential tests, but may proceed at once to treatment. In the more complicated cases, however, the urgency of the case is usually not so great but that a few days may be spent profitably in the carrying out of these differential investigations without detriment to the patient. With the exceptions of carcinomatous degeneration of a gastric ulcer; of pyloric obstruction due to cicatricial stenosis; of hour-glass constriction, sufficient to cause marked disturbance of gastric motility; of perigastric abscess, or of acute or chronic perforation, ulcer of the stomach or duodenum is a disease which lends itself to medical management in a majority of cases. But the medical treatment must be well directed, rigorous, long continued, and the best results are to be obtained where the patient is under hospital supervision; and in a hospital where there is cordial co-operation between the attending physician, the diet kitchen and the laboratory. If the case cannot be admitted to hospital control, treatment may be instituted at home, provided that the patient is attended by a competent trained nurse. The medical treatment of ambulatory cases is unsatisfactory to a degree, so far as ultimate cure is concerned, although suppression or amelioration of symptoms can usually be secured. This is strikingly seen in the dispensary groups of patients.

There are *three essential features* in the medical management: complete bed-rest, proper use of diets, and the administration of chemical therapy, notably the antacids and the antispasmodics.

The duration of medical treatment, of necessity indefinite, bears a somewhat direct ratio to the length of time the ulcer has been present. Acute superficial phagodenic ulcers, such

as are common to chlorosis, will usually heal in from three to six weeks' time, unless tissue repair has been diminished. Ulcers that have existed for a year or more, and have become chronic, in pathology as well as symptoms, may require several months of active treatment, with a careful following up for two or three years before a definite cure can be rightfully claimed. Some ulcers have become so surrounded by connective-tissue overgrowth, with indurated cicatricial edges, that their very devascularity prevents their ultimately healing. Such cases can only be cured by excision of the ulcer-bearing area, and this excision should be a wide one, as this type of ulcer is the one most likely to become cancer ultimately.

Before undertaking the medical management of ulcer cases there should be established a thorough co-operation between the patient and the doctor, and it is important that the length of complete bed-rest should be left an indefinite matter. Whether this will require three weeks or three months cannot be determined beforehand, and the patient will therefore be spared many disappointments and the doctor many fruitless arguments if this plan is adopted. The room chosen should be light, well ventilated, and as free from outside noises as possible. The bed should be of the usual high hospital type, and with a fairly hard mattress. After the patient has once been gotten to bed, bed-rest should thereafter be complete and absolute, even to the performance of all toilets, and the patient should rest in the recumbent posture with one, or at the most two, low pillows for at least the first two weeks, after which the partly sitting posture may be allowed.

If the ulcer should be complicated by a relative pyloric obstruction, due to a ptotic stomach with an angulated duodenum, and especially if further complicated by hypersecretion, more prompt emptying of the stomach may be secured if the foot of the bed is elevated 10 or 12 inches, and the patient is made to assume the right, or, preferably, the left, lateral abdominal position. In certain cases this may be reinforced by placing a small circular pillow, of the bolster type, at a point level with the greater curvature of the stomach, or proper supporting abdominal pads may be used for this purpose.

## GENERAL HYGIENE.

**Care of the Mouth.**—Particular emphasis is to be laid upon bringing the oral cavity and its contents to as high a degree of cleanliness as is possible. There is a very close association between various degrees of oral sepsis and the different diseases of the stomach, notably gastritis, ulcer and cancer, and a close inspection of the mouth should be made in patients about to be given treatment, and in those cases needing it diseased roots should be extracted, cavities filled, and pus pockets cleansed and evacuated by an expert dentist before beginning active treatment for the ulcer cure. A few days consumed in this way will be of inestimable advantage later on. In all cases the teeth should be properly brushed three times a day with any good tooth-paste, if there be any tendency to pyorrhea, and this should be followed by the thorough rinsing of the mouth and gargling of the throat with any one of several mild antiseptics, such as the liquor antisepticus alalinus. To insure oral asepsis, it is helpful to keep a tumbler of such a solution, properly diluted, by the bedside of the patient, and to encourage its frequent use. Gums that are definitely infected should be swabbed daily with diluted tincture of iodine, and pus pockets should be wiped out with either iodine or hydrogen peroxide, or with a solution of hydronaphthol, 5 grains to the ounce (0.32 Gm. to 30 mls) of equal parts of 75 per cent. alcohol and distilled water. This may be done once or twice a day. If amebæ can be demonstrated in the pus pockets, one may use emetin in  $\frac{1}{4}$ -grain (0.015 Gm.) doses, dissolved in 1 dram (4 mls) of water, and by means of a blunt needle injected directly into the pus pockets every day for four or five days, or they may be swabbed out with a weak solution of ipecac, 15 grains to the ounce (1 Gm. to 30 mls). If spirochetæ or spirillæ can be demonstrated in pus pockets they should be flushed out with a solution of salvarsan,  $1\frac{1}{4}$  grain (0.075 Gm.) dissolved in  $\frac{1}{2}$  ounce (15 mls) of normal saline solution. After the active infection and the inflammatory residue have subsided it is useful to massage the gums digitally, to re-establish the circulation of the gums and to promote healing. Especially is this care of the mouth indis-

pensable in those cases to be treated either by the preliminary starvation method or by duodenal feeding, in order to minimize the danger of parotitis. Suckling a rubber nursing-nipple, as recommended by Fenwick,<sup>11</sup> or chewing gum as recommended by Ochsner,<sup>12</sup> stimulates the flow of saliva, cleanses the ducts, and guards against an ascending infection of the parotid gland. The nipple should be kept in 95 per cent. alcohol when not in use.

Likewise, infected tonsils should receive appropriate treatment. Those cases which give a history of recurrent tonsillitis, with or without rheumatism or cardiac disturbance, and whose tonsils are notably diseased and are no longer capable of a *protective* function, should have a tonsillectomy done before the ulcer cure is actively begun. Milder grades of tonsillar infection should be locally attacked by antiseptic gargles and swabbing the tonsils with a silver solution in a strength of 10 per cent. to 25 per cent.

Where oral feeding is withheld, and the tongue becomes furred, thickened, edematous or dry, it will be found useful to swab the tongue and oral mucous membranes with the following solution: One ounce (30 mls) of glycerin and the juice of one lemon. Also, when indicated, appropriate local treatment should be given the nasal passages.

**Care of the Body.** Each morning there should be given a cleansing bath, either hot or tepid, always to be followed by a cold sponge, unless contraindicated, and this by an alcohol rub, and the body sprinkled with a talcum powder containing stearate of zinc. The cold sponge may be repeated again at night, if desired, and will prove helpful in inducing sleep; invariably it should be followed by the alcohol rub and the use of the powder.

In those cases who are undernourished, due to inanition, or in those where the skin is dry and scaly, a daily inunction of olive oil may be used. In patients who are much emaciated, or who show a tendency to bed-sore formation, the use of any of the various protective pressure rings or air pillows is indicated.

It is important to mention the usefulness of *proper breathing exercises* to increase the oxygenation of the blood, which the writer believes has a decided tendency to promote healing



of internal ulcerated areas. It will be of service to have the patient hold between the lips a quill toothpick whose pointed ends have been cut away, or any other suitable hollow tube of small caliber, and through this to take 20 to 30 deep inhalations and exhalations two or three times a day, either from an oxygen tank or by wheeling the bed near an open window.

**Local Applications of Heat or Cold to the Abdomen.** All cases, except those in which there has been a history of recent bleeding from the gastro-intestinal tract, should be treated by *hot* compresses, continuously applied for the first week to ten days of treatment. These hot compresses should be made of flannel folded into three or four thicknesses, or of spongiolin, either of which are to be cut to a size that will cover the entire abdominal surface from the xiphoid to the pubic bone, and should be wrung out of water as hot as can be borne. The compress should be covered by a layer of oiled silk and a snugly fitted abdominal binder, and both should be carried 1 to 2 inches (2.54 to 5.08 cm.) above and below the compress to prevent evaporation or cooling by outside currents of air. The abdominal binder should then be covered, preferably by an electric warm pad, or a partly filled hot-water bottle. These hot compresses should be changed every hour during the day and once or twice during the night. This use of moist heat will be found of great service for the relief of pain, and in allaying perigastric or duodenal inflammation, or a localized peritonitis, and may even have a tendency to soften and separate *recent* perigastric adhesions. Where there is sufficient inflammatory perigastric exudate to cause a palpable tumor, especially in the presence of a leukocytosis, hot flaxseed poultices should be made to alternate with the hot compresses, and changed hourly, or every half-hour, until improvement is noted. Before beginning the use of these hot applications, it is well to sterilize the skin to avoid any possibility of infection from blister formation. For this purpose the writer prefers the solution recently recommended by A. D. Whiting.<sup>13</sup> After extensive tests it has shown the highest efficiency. The formula consists of acetone, 35 mils; phenoco, 2 mils; alcohol (95 per cent.), q. s. ad 100 mils. Rub the skin for two minutes with a piece of gauze saturated

with the solution. The sterilization should be repeated on the second and third day and then discontinued.

Where there has been a history of recent gastro-intestinal hemorrhage within from six weeks to two months before beginning treatment, or where occult blood can be demonstrated in the stools, the *use of hot compresses is contraindicated*, and extreme degrees of cold are to be substituted, in the form of constant applications of ice-bags, particularly to the epigastrium, or the use of ice-water coils in hospitals suitably equipped. The use of cold, rather than heat, should be continued for one week, or until all traces of altered blood have disappeared from the stools, when the use of hot compresses should be begun as outlined above.

When there has been recent hemorrhage, no food or drink should be allowed by mouth until all occult blood has disappeared from the stools.

Everything should be done to insure complete mental and physical relaxation, to protect the stomach from insult, mechanical, chemical and thermal, to provide sufficient nourishment to keep up the bodily vigor of the patient, to prevent undue immediate loss of weight, and later to promote a gain in weight.

Foods should be bland, furnished in a proper form, and in only sufficient amounts at any one time as will not encourage mechanical overactivity of the stomach or exert a drag on its supporting ligaments. As a rule, it is wise to insist upon total food abstinence by mouth in all cases, and certainly in those of the bleeding type, for a period of from three to seven days, the time usually averaging five days. During this period especial attention is to be directed to the care of the mouth, as outlined above.

The most disagreeable subjective feature during this time is a somewhat excessive thirst. This is to be controlled by cleansing the mouth and by sucking cracked ice, care being taken not to swallow any. The chewing of gum, as pointed out by Ochsner, helps to relieve thirst. The saliva may be swallowed. In addition, the administration of various liquids by rectum is indicated. In the writer's opinion the best is a decinormal solution of soda bicarbonate, rather than decinormal salt solution, inasmuch as it has a tendency to combat

an acidosis arising from starvation. Stronger concentrations of soda bicarbonate are not necessary; they may be unduly irritating, and have a tendency to cause increased intestinal peristalsis, which is something to be avoided. It may be permissible to add to the decinormal soda bicarbonate solution sufficient glucose in percentages ranging from three to five, remembering, however, that sugar and albuminous substances tend to promote putrefaction in the bowels. It is best to administer this solution by the Murphy method, adjusting the rate of the drops so that about 500 mils (16.67 oz.) can be introduced in an hour. Should this rate of flow cause discomfort, it should be decreased to the point of easy tolerance. The rectal tube should not be introduced farther than from 3 to 5 inches (7.62 to 12.70 cm.). As a rule, it is not necessary to give more than a total of 2000 to 2500 mils (66.67 to 83.84 oz.) of this solution in twenty-four hours. In the event of hemorrhoids, they should be treated by cold compresses soaked in the fluidextract of hamamelidis, and internal hemorrhoids by the application of a 5 per cent. ointment of extract of hamamelidis with equal parts of lanolin and petrolatum, introduced by means of a hemorrhoidal syringe. This treatment should follow the enteroclysis and the use of rectal alimentation.

**Nutritive Enemas.** It is the custom of many to make use of nutritive rectal enemata during the period of all food abstinence, but it is doubtful whether this is actually necessary as a supportive measure, and in the writer's experience it frequently causes so much discomfort to the patient as to neutralize its possible beneficial effect. There are many standard nutritive enemas to which reference is made in the monographs on the subject, but the writer has had most satisfaction from the following formula: Milk 200 mils (66.67 ozs.), the yolk of one egg, milk-sugar 15 grammes (231.45 grs.), to which is added a pinch of salt. This should be thoroughly stirred and peptonized for ten minutes by adding the contents of one of the tubes of Armour's or Fairchild's pancreatic powder; after this the mixture should be injected very slowly at a temperature of 110°, and with the patient's hips slightly elevated. If the rectum is irritable, a few drops of the tincture of opium or of the deodorized tincture of opium

may be added to the enema, three or four of which may be given during the course of twenty-four hours.

**General Medicinal Management.** Except as needed to control urgent symptoms, the use of chemical therapy does not have anything like the importance in the medical cure of ulcer as does complete bodily and mental rest, proper feeding, and external applications to the abdomen. *The use of drugs should be withheld, unless their use is definitely indicated.*

The writer knows of only three drugs which may directly aid in the healing of an ulcer. In an acute ulcer, without indurated edges, the use of bismuth acts not only as a mild antacid, but serves to coat over the floor of the ulcer and partly to protect it from the corrosive action of the gastric juice, and irritation from food particles.

Where bismuth is used the subcarbonate is the best form, and may be given in doses of from 10 to 20 grains (0.65 to 1.3 Gm.) every two to four hours. The subnitrate of bismuth should not be used on account of the danger of mechanical irritation by its sharp crystals.

In the chronic, sluggish, indolent ulcer the use of silver nitrate may serve to stimulate the formation of granulation tissue, and to promote healing. Certainly, it can be said that in ambulatory cases of ulcer of this type subjective improvement is seen by the use of lavage with a solution of silver nitrate of a strength beginning with 1:5000, and gradually increasing to a strength of 1:1000. Where silver nitrate is given orally, the writer prefers to employ the nine-day cycle suggested by Lockwood and described on page 708.

The third drug of essential service is belladonna, or its alkaloid atropin. Its usefulness lies in the fact that it most effectively controls gastric secretion and lessens its concentration, and, furthermore, it retards hyperperistalsis and diminishes gastric motility. The rationale of its use is therefore plain, and, besides, it is well tolerated. It should be administered in the form of the tincture of belladonna, given every four hours, beginning with 5 minims (0.30 Gm.), and increasing 1 minim (.06 Gm.) each time taken, until a definite physiologic effect is produced, namely, a dilatation of the pupils, with blurring of vision, and a dryness of the mouth and fauces. When this point is reached, which will usually



occur with a dosage of 15 minims (1 mil), the drug should be discontinued for one or two doses, and then be continued in a constant dosage of 3 to 5 minims (0.1 to 0.3 mil) less than the dose producing physiologic effect, and maintained at this point for two or three weeks.

Where there is hypersecretion or hyperacidity, the use of alkalis becomes absolutely necessary. In addition to bismuth and belladonna, the following drugs may be recommended: magnesia usta (the oxide of magnesia) and sodium bicarbonate. These may be combined in powder form with bismuth subcarbonate in equal parts, in a dosage of 10 to 20 grains (0.6 to 1.2 Gm.) given every two to four hours, suspended in water.

Bolton<sup>14</sup> urges the use of lime-water in place of soda bicarbonate, because the latter, in neutralizing hydrochloric acid, does so with the liberation of carbon dioxide gas, which distends the stomach unless promptly eructated. Furthermore, Bolton believes that lime-water does not excite gastric secretion. He gives lime-water in teaspoonful doses about midway between each feeding.

Working on the hypothesis that hyperacidity is one of the potent influences in retarding the healing of an ulcer, Sippy has recommended the following plan of administering alkalis. He enjoins absolute bed-rest, and for five days withholds all food and drink by mouth, and begins hourly feedings on the morning of the sixth day. Each morning, one-half hour before the first feeding, he gives 1 teaspoonful (3.75 Gms.) of bismuth subnitrate, suspended in one-half glass of water, and midway between each feeding he gives, alternately, a powder suspended in 1 ounce (30 mls) of water, consisting of calcined magnesia 10 grains (0.6 Gm.) and sodium bicarbonate 10 grains (0.6 Gm.), and a second powder of bismuth subnitrate 10 grains (0.6 Gm.) and sodium bicarbonate 10 grains (0.6 Gm.). Thus the patient receives 20 grains (1.2 Gm.) of an antacid powder every hour, midway between feedings. If the powder containing magnesia produces diarrhea, this is to be prevented or controlled by substituting the powder containing bismuth a sufficient number of times to control it, and, conversely, if constipation ensues, the magnesia and soda powder should be given more frequently.



After the fourth or fifth week, as the amount of food is increased and the time interval lengthened, the powders are still to be given midway between the feedings, but "the quantity taken each time may be proportionately increased."<sup>15</sup>

Sippy continues the use of the morning bismuth, in teaspoonful doses, for six or eight weeks, and continues the use of the other powders, midway between the feedings, for three or four months if the ulcer is a recent one, and intermittently for several months longer if the ulcer is of long duration.

As a routine procedure in all cases of ulcer, this method may be open to criticism, and should be reserved for those cases of proved hyperacidity, with or without symptoms, and, if adopted, the writer prefers the use of the subcarbonate of bismuth to that of the subnitrate, for the reasons stated above. (See p. 701.)

Of other alkalies, Carlsbad water has long enjoyed the reputation of being a truly medicinal agent in the cure of ulcer. This view is particularly held by European clinicians, and is especially endorsed by von Leube. It is particularly useful in the constipated cases, and may be given in  $\frac{1}{2}$ - to 1-glassful doses, once or twice a day on a fasting stomach. It is not always possible to obtain the original Carlsbad water, and the desiccated salts may be substituted in a dosage of 15 grains (1 Gm.) to a glassful of water. Probably the artificial Carlsbad salts, the *sal carolinum factitium* of our pharmacopœia, are equally good, easier to obtain, and cheaper. The dosage should be 1 teaspoonful (3.75 mls) of the salts in a glassful of water once or twice daily.

Many other alkaline waters may serve a similar purpose, and the writer particularly likes to make use of Celestins Vichy, and especially during the later weeks of the illness, and in the follow-up plan of treatment.

Paul Cohnheim is an enthusiastic indorser of the use of olive oil. He recommends the use of 2 to 4 drams (7.5 to 15 mls) of pure olive oil, three or four times a day before feedings, in those cases exhibiting the pain of pylorospasm, and the use of an oil emulsion, 1 teaspoonful (3.75 mls), of either olive or almond oil, the yolk of one egg, and water to

make 3 ounces (90 mls), to be used in cases without pylorospasm. The writer believes that the use of oil does not facilitate the healing of an ulcer, and confines its use to such ambulatory cases as will neither submit to a thorough medical *régime*, nor consent to operative interference.

**Diet.** The essential features of the diet are threefold: (1) to provide sufficient nourishment; (2) to be of small bulk and of a bland, non-irritating type; and (3) to counteract or control hyperacidity. This last is an especially important essential.

For many years only two methods of feeding were advocated in ulcer cases, that of von Leube and that of Lenhartz, and between the two a controversy existed which continued for years, and which has been kept alive by the different followers of these gastric clinicians, first in Europe and later in this country. These two methods differ rather widely one from the other, but that of von Leube has proved more acceptable to the general clinician. The character of this article does not permit of an extensive review of these two methods, which are quoted in detail in many monographs, but they differ essentially in these points: the von Leube method consists in withholding all food by mouth, in all cases, for a period of three days, with the patient under complete bed-rest for ten days, and then proceeding to the use of increasing amounts of food each day. The diet is of a type that is bland and non-irritating, and largely composed of carbohydrates, which tend to diminish both the amount and concentration of the gastric juice. By the Lenhartz method one proceeds to feed the patient at once, even in the face of hemorrhage, using repeated small feedings of a variety of highly albuminous foods, selected for their ability to combine with the free hydrochloric acid of the gastric juice so as to form a loosely combined acid albumin, which Lenhartz believes prevents further erosion and facilitates healing. He maintains that the recuperative forces of the patient are depleted by the under-feeding of von Leube's method, and that the tendency of the ulcer to heal is thereby retarded.

It is by no means necessary strictly to adopt either one of these two methods in the exact detail of its originator, nor does the writer believe that every case can be religiously

treated by the same dietetic method.\* As a general rule, the principle of placing the stomach in a state of complete physiologic rest for a few days is a sound one, and this can best be accomplished by abstaining from all food by mouth for three, or not more than five, days. Yet the use of rectal enemata, and in certain cases even the use of proctoclysis, excites intestinal peristalsis, which, in turn, may reflexly produce pylorospasm and gastric peristalsis, and thus increase the difficulty of securing physiologic rest. Again, there are some profoundly cachetic patients in whom it may be expedient to begin feedings at once, barring recent hematemesis. In such instances, rather than run the risk of undermining further the patient's vigor and recuperative power by total food abstention for even two or three days longer, it seems better to dispense with the preliminary fast.

In all cases the writer proceeds to feed at once, using a formula personally communicated to him by Joseph Sailer, and as yet unpublished. This formula consists of cream, whites of eggs, lactose and rice-water. The caloric strength of this mixture can be readily augmented by gradually increasing the amounts of the first two ingredients, and by adding later the yolks of eggs. This formula is bland and rich in albumin, and the writer has yet to see a case in which it is not easily tolerated by even the most sensitive stomachs. For the *first day's* feeding it is well to begin with the following: cream, 2 ounces (60 mls); white of 1 egg; lactose, 1 ounce (30 Gms.), and rice-water sufficient to make 1 pint (473.11 mls). The rice-water is to be made by thoroughly boiling 1 ounce (30 Gms.) of clean rice in a pint (473.11 mls) of water. The value of this formula is approximately 300 calories. This, a rather flat, tasteless mixture, may be made more palatable by the addition of 4 ounces (120 mls) of chocolate or cocoa, prepared in the usual manner, or an equal quantity of black coffee; or sufficient grape-juice may be added to make it

---

\* The reader is referred to a recently published article by Smithies (Smithies: Am. Jour. Med. Sc., 1917, cliii), who calls attention to the good results he has obtained in the treatment of gastric ulcer by a carbohydrate diet and abstaining as much as possible from the use of antacid medication. This paper appeared too late to be incorporated in this chapter.

palatable. This mixture should be bottled and packed in ice, and should be served very cold, beginning with 2 ounces (60 mls) every hour from 7 A.M. until 7 P.M. Patients should not be disturbed for feeding at night more than once or twice, and the food should be given at the same time that the hot compress applied to the abdomen is changed. The total caloric value for this first day will be approximately 500. This formula feeding should be continued for the first seven days.

In feeding all liquid foods that are to be served cold, it adds much to the comfort of the patient and the acceptability of the food, if care is taken to have it attractively served and ice-cold. The 2-, 4- or 6- ounce (60, 120 or 180 mls) glass, according to the amount of each feeding, should be of a thin glassware, and placed in the center of a deep bowl, such as a finger-bowl, packed with crushed ice; the spoon, also, should be kept well iced.

If there is no discomfort arising from this first day's feeding, on the *second day* the formula may be strengthened by doubling the amount of cream and egg albumin and feeding in 2-ounce (60 mls) doses, every hour from 7 A.M. until 7 P.M., and one or two feedings at night. This will represent approximately 900 calories.

The *third day* the formula may be strengthened by using 2 whole eggs, whites and yolks instead of the whites alone, and the feedings may be increased to 3 ounces (90 mls) every hour from 7 A.M. until 7 P.M., with one or two feedings at night. This will approximate 1500 calories. Feedings should only be increased from 2 to 3 ounces (60 to 90 mls) in amount, provided there has been no discomfort from the second day's feeding. As a rule, under this dietetic *régime* all pain and gastric discomfort promptly subside. Occasionally, however, even this amount of food produces an increase of gastric tension, or the painful sensation of pylorospasm. In such an event, it is well to abstain from all foods by mouth and to adopt the methods outlined above. In any case it is advisable, however, to give proctoclysis to the extent of 2 quarts (2 l.) of decinormal soda bicarbonate solution, inasmuch as this will tend to combat the development of acidosis, which may arise from persistent feeding of a diet rich in butter fats, a possibility to which Pritchard has drawn



attention. The hot compresses are to be applied and changed every hour during the day, and once or twice during the night, as outlined above, during these first seven days. The bowels should be moved each day with a cleansing enema, except in those cases in which the three-day starvation plan is adopted, when the bowels are to be moved by a cleansing enema the night before beginning treatment, but need not be moved again until the third or fourth day. Should tympanites develop during this first week, usually it can be relieved by substituting turpentine stupes for the hot compresses, and by using the rectal tube. Should this not suffice, hypodermic injections of eserine sulphate in a dosage of  $\frac{1}{50}$  grain (0.001 Gm.) may be given every three hours for three or four doses. No drugs are to be administered by mouth, with the possible exception of soda bicarbonate, 10 or 20 grains (0.6 to 1.2 Gms.) of which may be dissolved in 1 or 2 ounces (30 to 60 mls) of water, and given every second hour between the feedings. It is usually wise to determine the presence or absence of free hydrochloric acid in the stomach by the use of a duodenal catheter, introduced between or just before feedings. Should free hydrochloric acid be present in any degree above 15 acidity per cent., the soda bicarbonate solution may be resorted to; otherwise chemical therapy should be entirely withheld, except where it is necessary to combat certain symptoms, such as pylorospasm, hyperacidity or hypersecretion, which will be described later.

On the *fourth day* the amount may be increased to 5 ounces (150 mls) every two hours, with one or two feedings at night, and on the *sixth* and *seventh* days may be still further increased to 6 ounces (180 mls) every two hours, with one or two feedings at night. Should this formula in any wise disagree, peptonized milk may be used alternately every other feeding in the amounts scheduled for that day. This will, however, somewhat reduce the caloric value. Lockwood's<sup>16</sup> method of peptonization is an excellent one, and is as follows:

"To 1 pint of milk there should be added  $\frac{1}{4}$  pint of water, and the mixture is to be divided into two equal parts. Boil one part, and immediately afterward add the other. Stir in the contents of a peptonizing tube, and set the bottle in warm



water for one and one-quarter hours. Bring rapidly to a boil and keep on ice. The completely peptonized milk should have a slightly bitter, but not unpleasant taste."

**Treatment from the Eighth to the Fifteenth Day.** The two-hourly plan of feeding should be continued during the day with only one feeding at night, which is to be given at midnight, but there may be substituted for any one of the formula-feedings one of the following articles in amounts not to exceed 6 ounces (180 mls): mammala; any cooked cereal, such as cream of wheat, farina, wheaten, oatmeal or arrow-root gruel may be eaten with cream and sugar; creamed macaroni or spaghetti, without cheese; boiled rice, milk toast, soft-boiled egg, egg-custard, blanc mange, junket, vanilla ice-cream, wine-jelly or calf's-foot-jelly.

The eighth day only one substituted article is to be given. The ninth day two may be permissible, and for the balance of the second week three substitutions may be allowed. Every attempt should be made to vary the diet so as to avoid monotony, and to cater especially to the patient's taste.

The external applications are to be continued during this second week, but need be changed only every second hour. Should this increase of diet during the second week be provocative of any symptoms, such as discomfort due to increased acidity, pylorospasm, or the recurrence of occult bleeding, the dietary should be reduced to that of the first week. The daily cleansing enema is to be continued. In those who are unduly constipated or flatulent, and especially in those patients with furred tongues, undue drowsiness, headache or other evidence of hepatic torpor, the use of Carlsbad water is indicated, or the artificial salts *sal carolinum factitium* may be substituted, using 1 level teaspoonful (3.75 Gms.) dissolved in a glassful of water. This should be given cold to those whose constipation is of the atonic type, and hot to those having spastic constipation. Two or three doses may be given each day according to the results obtained. The first dose should be given on an empty stomach one hour before the daily feedings are begun. This, likewise, will tend to counteract any hyperacidity. Equally good results, however, may be obtained by the use of magnesia, either alone as the milk of magnesia in teaspoonful (3.75 mls) doses two or three times a day, or the

oxid of magnesia, 10 grains (0.65 Gm.), to be combined with a like amount of soda bicarbonate, and to be dissolved in from  $\frac{1}{2}$  to 1 glassful of water. In those cases in which pain persists, notwithstanding the above measures, the use of silver nitrate, according to the plan suggested by Lockwood,<sup>17</sup> may be adopted. He recommends the following prescription:

R Argenti nitratis ..... gr. 16 (1.03 Gm.).  
 Aqua destillata ..... ℥ij (60 mils).  
 M. Sig.: Five minims equals gr.  $\frac{1}{6}$  (.01 Gm.).  
 Give 15 to 25 minims (0.9 to 1.5 mils) in distilled  
 water, thrice daily, one-half hour before eating.

Lockwood recommends using this in nine-day cycles as follows: The first three days give 15 minims, which equals  $\frac{1}{2}$  grain (0.0325 Gm.), three times a day; the second three days give 20 minims or  $\frac{3}{4}$  grain (0.04875 Gm.) three times a day; for the next three days give 25 minims or  $\frac{5}{6}$  grain (0.05416 Gm.) three times a day. Any resulting diarrhea should be met by reducing the dose or entirely withholding it. Lockwood states that "the silver nitrate cycle seems to be indicated especially in those ulcers, with clean tongues and regular bowel functions, which are accompanied by a heightened acidity, and usually with persistent pain." During this first fourteen days absolute rest in bed should be strictly enforced.

#### Treatment from the Fifteenth to the Twenty-first Day.

The formula feedings may be reduced to two or three a day, taking 8 ounces (240 mils) in the middle of the forenoon, the middle of the afternoon and at bedtime, and the feeding during the night may be discontinued. In those cases in which milk-foods are not well borne, the formula feedings may be discontinued, and the articles allowed during the second week may be increased by the addition of cream soups or purées, except those made from meat or meat-stock, which excite too strongly the secretion of the gastric glands; mashed, creamed or baked potato, if thoroughly cooked; zwiebach or crackers, which must be thoroughly masticated. Creamed fish and all vegetables that can be mashed and put through a colander and served in a soft purée, such as squash, cauliflower-tips, spinach, turnips, and soft desserts, as tapioca, rice-pudding and floating island are permissible. Two or three of these

articles of diet may be given at one time, and the feedings should be given every three hours. During this third week the patient may be allowed to rise for the essential toilets. The external applications should be continued according to the plan of the second week. The bowels should be kept regulated by enemas and the use of the Carlsbad treatment. With the increase of diet allowed during the third week, the gastric secretions may be so stimulated as to need the use of antacids, such as soda bicarbonate, magnesia or lime-water, which may be combined with bismuth subcarbonate in a powder form, consisting of the oxide of magnesia, 10 grains (0.6 Gm.); bismuth subcarbonate, 10 grains (0.6 Gm.); soda bicarbonate, 20 grains (1.2 Gm.). This is to be dissolved or suspended in water, 2 or 3 ounces (60 or 90 mls), and to be taken one-half hour after meals. If there is gaseous distention, due to fermentation, 1 grain (0.065 Gm.) of creosote may be added to this powder. If the bowels become too loose, the magnesia should be withdrawn. It is well to determine the necessity of the use and the dosage of these antacids by ascertaining the acidity of the gastric content at some time between or toward the end of each feeding, since it is their purpose to neutralize the excess free hydrochloric acid, and thereby facilitate healing.

During this third week anemia may be combated by the intramuscular injection of sodium cacodylate and the citrate of iron, beginning with 1 grain (0.06 Gm.) of each every second day, and gradually increasing the dose to 3 grains (0.2 Gm.).

**Treatment from the Twenty-second to the Twenty-eighth Day.** The patient may now be allowed to sit in a comfortable chair for several hours each day, and may be wheeled to sun-parlor or grounds, but should be made to lie down for one hour after each feeding. The external applications of heat may now be reduced to such times as the patient is in bed. The menu may be increased by the addition of such articles as creamed sweetbreads, creamed chicken, turkey, guinea-hen, squab, broiled white fish, and tender chopped veal. Soft peas, tender string-beans, bread and butter, dry toast, chocolate and cocoa are useful. Otherwise the plan of the third week is to be continued. Should the appetite be diminished, strychnin

or nux vomica may be prescribed in some simple stomachic vehicle, such as the compound tincture of gentian or the compound syrup of hypophosphites.

**The Follow-up Treatment.** In no disease should the follow-up treatment call for closer understanding and co-operation between the patient and the doctor, and no complete medical cure can be expected unless this is so. *Many ulcers, which are well on the way toward permanent healing, later relapse and require surgical interference for ultimate cure, as a rule, only because sufficient emphasis had not been laid on the after-treatment.* Every doctor is totally remiss in the responsibility he owes his patient if he does not make this part of his method of treatment thorough and long sustained, and the patient is glaringly guilty of thoughtlessness and indifference to the best interest of his health, if he does not realize the wisdom of the instructions given him.

During the fifth and sixth weeks of the cure the plan of the fourth week is to be maintained, and especially is this true regarding the continuance of bodily and mental rest. As much of the time as possible should be spent lying down, or pursuing any gentle occupation that does not entail physical exertion. Usually after the sixth week partial resumption of business duties or household affairs may be begun, and moderate exercise may be resumed, at first in the form of walking only. Later golf is permissible, at first a few holes, and never more than eighteen, may be played on any one day. Games requiring more violent exercise, such as handball, boxing, tennis, rowing, swimming, horseback riding, polo, and the like, must be postponed for at least six months. Plenty of hours should be devoted to sleep, and at least ten to twelve hours of the twenty-four should be passed in the recumbent position.

Patients should be urged to lie down for at least one-half hour after each meal. During the period of physical inactivity the muscles and skin may be kept in a state of good tone by the use of electricity or hydrotherapy. In certain cases, especially those constipated, this will prove of exceptional advantage, and for sluggish bowels the Bergonie method of electrical treatment is to be recommended. The bowels should be kept well opened during the period of physical inactivity, to insure which they need to be aided by the occasional use

of simple enemas, or the use of liquid paraffin, Carlsbad or other mildly laxative alkaline waters; or the use of magnesium oxide in powder form, combined with soda bicarbonate, in a dosage of 5 to 10 grains (0.324 to 0.65 Gm.) each, to be given after or between meals, at the high-point of gastric secretion, as determined by fractional analysis; if preferred, the milk of magnesia, in teaspoonful (3.75 mls) doses, may be substituted for this purpose. If it is found that the gastric secretions continue too highly acid or in increased amounts, it is advisable to return to the use of silver nitrate as detailed above, or atropin or its derivatives may be administered, subcutaneously or by mouth, to a point of physiologic effect. If the gastric analyses show hyperacidity, antacids such as recommended above should be used. If hypersecretion continues the stomach should be emptied night and morning, by means of a gastric or duodenal catheter and a small aspirating syringe. Most patients become readily accustomed to this form of tube, and many of them prove capable of doing this for themselves. As to general hygiene, especially as regards the care of the mouth, teeth and skin, patients who have undergone active treatment will prefer to continue the use of such toilets indefinitely, but, nevertheless, it should be made a part of their instructions.

The use of alcohol in all forms, and especially the highly concentrated ones, such as cocktails, should be prohibited for at least six months, with the exception of a glass or two of a good claret, or Rhine wine, or the use of rye or Scotch whisky, occasionally, in the form of a highball with plain water, for those who are accustomed to its use. This will probably do them no harm, but it is wiser to urge total abstinence. All effervescent or aerated drinks should be prohibited. Free use of a mild alkaline water, such as Celestins Vichy, should be encouraged. Smoking should be abstained from for at least six months, with the exception of the occasional use of a mild cigar after meals. Tobacco in any other form, especially chewing, should be prohibited.

Finally, no part of the after-treatment is more important than the adherence to a proper diet, for at least six months to a year, after the period of active treatment. In general, all food that stimulates gastric secretion or causes hyperperis-



talsis, should be entirely interdicted. This will include all the spices and condiments, such as paprika, pepper, mustard and horse-radish; salt should be used sparingly; likewise sauces, such as tomato catsup, chili, Worcestershire and Oscar sauces, or the use of such vegetables as onions, radishes, leeks, cucumbers and tomatoes; or pickles, mixed pickles, and acid fruits of the citrate family, cherries, bananas, and the like. All such articles of food should be forbidden. Fresh fruits should be indulged in only sparingly, and it is better to give preference to the cooked forms. Likewise, all fried foods, hot breads and pastries, all rich dressings, gravies and sauces, or rich desserts, and all meats tough with connective tissue, and scratchy vegetables with outer shells of cellulose, such as corn and celery, should be abstained from for at least a year, if possible. Emphasis should be laid upon the necessity of thorough mastication. Great care, too, should be taken not to overload the stomach at any one time. This applies to both liquids and solids, and it is wiser to adopt a five- or six-meal plan of feeding in small amounts. Very hot or very cold foods should not be taken. The patient should make it a practice to lie down for at least one-half hour after each meal.

As to what may be eaten, the writer recommends the use of the following menu, which has served him well in the past:

*Breakfast.* A cooked cereal, such as farina, wheatena, cream of wheat or hominy may be eaten with cream and sugar. Oatmeal may be allowed, if very thoroughly cooked. An occasional lamb-chop or slice of breakfast bacon. Two soft-boiled or poached eggs. The soft parts of bread, crackers or freshly made toast may be eaten with butter. Milk, malted milk or cocoa may be taken. It is better to avoid both tea and coffee, although they may be used for flavoring.

10 to 11 A.M. The choice of the cream and rice-water formula, malted milk, koumiss, keffir or buttermilk, or equal parts of milk and cream, junket or cup-custard. One or two raw eggs may be substituted or added to any of the foregoing. Crackers and butter.

*Luncheon or Dinner.* Chicken or fish in any form but fried, broiled squab, or the breast of guinea-hen. Broiled or boiled beef and lamb, to be run through a grinder when cooked. Milk-toast. Oysters in any form but fried. Potatoes in any

form but fried, preferably mashed or baked. Peas, lima beans, spinach, squash (to be put through a colander and puréed with cream), boiled rice, tender string-beans, buttered beets, creamed carrots, or the tender ends of asparagus and cauliflower, spaghetti or macaroni. A salad of plain lettuce with French dressing (with the amount of vinegar reduced) may be permissible every second or third day if desired. Bread and butter. Choice of junket, cup-custard, blanc mange, tapioca, rice, cornstarch or bread-puddings, floating island and vanilla ice-cream (if held in the mouth and warmed to body temperature).

4 to 5 P.M. The same choice as at 10 A.M.

*Supper or Dinner.* Thick soups, such as rice, sago, barley, farina, potato or asparagus, or creamed purées of beans, peas or lentil, which are to be run through a colander. No soups made from meat or meat-stock are allowed. One or two soft-boiled or poached eggs. Bread and butter. Milk or cocoa, and the choice of any of the above desserts, except ice-cream.

Before retiring, the choice of the foods allowed at 4 P.M.

During the course of this after-treatment the patient should be in personal or written communication with his physician, preferably the former, every two weeks for a period of at least six months, preferably a year, so that the earliest signs and symptoms of an exacerbation can be detected promptly. During this period a physical examination, to include particularly an inspection of the mouth and teeth, an abdominal examination, and routine analyses of gastric secretion and chemical analyses of the stools, should be made at least *once a month*. It is the writer's firm belief that if this plan of medical management of ulcer cases is adopted in its entirety, a larger number of real medical cures will be secured, with a consequent lessening of the number of patients who otherwise would require operative interference. Unless there are already existing complications or sequelæ of gastric or duodenal ulcer, as outlined above, every case should be given the benefit of medical treatment before resorting to surgery. This applies particularly to the border-line group. No medical plan of management, however, can be relied upon to secure results, unless it is thoroughly planned and rigidly adhered to, and every physician should devote as much pains-

taking care in the working out of the detail of his technic as that which pervades the operating room of every thoroughly capable surgeon. If the medical *régime* cannot, for one reason or another, be thoroughly carried out, the preference should be given to the aseptic scalpel, but only when wielded by the hand of the experienced full-time surgeon. If there is one thing worse than poor medicine or medical management, it is poor surgery.

There is a certain number of ulcer cases that, even with the best of medical handling, will relapse in the sense of giving signs and symptoms of ulcer activity. In these cases no further expectation of medical cure can be hoped for, and the case then becomes amenable only to surgical interference. If the case is uncomplicated to begin with, one will rarely see the development of complications under this medical plan.

**Method of Duodenal Feeding in Gastric and Duodenal Ulcer.** In recent years there has been a larger number of cases of ulcer treated by a plan first suggested by Einhorn, nearly a decade ago, and since modified by many. This plan aims at short-circuiting the stomach and duodenum and the establishment of physiologic rest, in the sense that no food is to be allowed to pass across the ulcerated area. This is supposed to be accomplished by the use of a duodenal tube or catheter, of a type first devised by Einhorn, and later modified by Rhexuss and others, which is passed, or swallowed by the patient, until it reaches a point in the second portion of the duodenum below the point of ulceration. This point may be determined, in many cases, by the string test if the ulcer is a bleeding one, or by fluoroscopic or röntgen plate analysis when interpreted by an expert. After this point has been determined and the duodenal catheter has been passed, it is an essential that a second fluoroscopic examination should be made to determine that the metal tip lies in the duodenum beyond the point of ulceration. For this form of treatment to result successfully, it is almost a necessity that the patient should be in a properly equipped hospital, since it is often wise to refluoroscope the patient every few days, to make sure that the end of the tube has not been regurgitated back into the stomach. This occurrence, which is not uncommon in

the writer's rather limited experience with this method, will entirely negate any possibility of success.

The duodenal tube should be at least 1 meter (39.37 in.) long and made of good rubber with a caliber of about 3 millimeters (0.12 in.), and should be marked at distances of 55, 70 and 80 centimeters (21.649, 27.559 and 31.496 in.) from the capsule end. The first mark represents the distance from the incisor teeth to the greater curvature of the stomach in the average case. The additional distance of 15 centimeters (5.866 in.) will usually carry the tube well into the duodenum, and it should never be allowed to pass further than the third mark. If there is an associated gastropnoxis an allowance of distance should be made according to the position of the stomach.

The metal tip should be of a kind that will not corrode by contact with the gastric juice, and provided with slots or perforations which are equal in caliber to the lumen of the tube. It should be made the rule *always* to provide fresh rubber tubing for each patient so treated, and to see that the metal tip is properly and securely tied into the tube with a fine surgeon's silk.

The tube may be either introduced through the mouth or through one of the nostrils, and should, preferably, be passed at night. If fluoroscopic facilities are not at hand to confirm the proper position of the metal capsule, the following points may be carried out: First, the tube should have reached the second marking, or a point between the second and third marking; second, when gentle traction is exerted a sense of resistance and tautening of the tube will be felt; third, by gentle aspiration there should be recovered a material that, in color and chemical examination, should conform to that of the duodenal juice; and, fourth, water, or preferably milk, when swallowed cannot promptly be aspirated through the tube. When the tube has reached the desired point it should be securely held in place by strapping with adhesive plaster, carried across the cheek, with the proximal end of the tube adjusted back of the ear, and fastened to the ear by a rubber band; when not in use the tube should be properly clamped off.

The foods given must necessarily be liquids, and Einhorn

recommends the use of milk, lactose, and raw eggs in the proportion of 1 glass of milk, 1 egg, and 1 tablespoonful (15 mils) of lactose. This should be stirred thoroughly and heated very slowly to avoid lumpiness, due to coagulation of egg-albumin, and should be injected very slowly every two hours from 7 A.M. to 9 P.M., at a temperature of from 100° to 105° F. (37.5° to 40.5° C.). The amount of each feeding should at first be 100 mils (3.34 ozs.), gradually increasing to 250 mils (8.34 ozs.). Amounts larger than this are likely to cause increased duodenal distention, even if given slowly.

Later, feedings may be given at intervals of one and a half hours, so that the total caloric value approximates 2500 to 3000 calories. Before each feeding about an ounce (30 mils) of sterile salt solution, or preferably, decinormal solution of soda bicarbonate should be introduced through the tube, and following each feeding a similar procedure should be carried out, and this followed by the injection of a syringe-barrelful of air. This serves to cleanse the tube of the feeding formula, and to keep it clean, which is of the utmost importance. If abdominal distention or flatulency occur, the feeding formula should be peptonized with pancreatic powder. The writer prefers the method suggested by W. G. Morgan, of placing the proper amount of the feeding formula in a graduated glass tank and feeding by a drop-method, similar to the Murphy method of proctoclysis, with the rate of flow so adjusted that it will require fifteen minutes to introduce each 100 mils (3.34 ozs.) of the mixture.

In patients who are considerably emaciated Smithies recommends the feeding of a mixture of amino-acid and maltose. This mixture is prepared by "first digesting Witte peptone in normal salt by trypsin (Fairchild) under toluol, or in 0.4 per cent. alphazone-normal salt solution, and then adding sufficient maltose to make a 5 per cent. mixture." He gives amounts of from 100 to 500 mils (3.34 to 19.99 ozs.) of this mixture through the duodenal catheter every three hours, and if thirst is excessive he gives an equal amount of normal salt solution about ten minutes before the amino-acid-maltose mixture.

These duodenal feedings may be kept up for a period ranging between fourteen and twenty-one days; shorter



periods than this cannot be expected to make much headway in the healing of an ulcer. During this period it may be wise to reinforce the duodenal feedings with a nutrient enema each day, and the use of decinormal soda bicarbonate in the dosage previously described. After the second or third week of treatment the duodenal feeding should differ in no wise from that which is recommended in the foregoing paragraphs. Complete bed-rest, hygiene, external applications of heat, and the follow-up plan of treatment should be just as energetically carried out.

As a rule, in patients who become tolerant to its presence it is not necessary to remove the duodenal tube until the desired time limit has expired, if great care is practised in keeping the tube cleansed after each feeding. After the tube has been permanently withdrawn, feedings by mouth may be resumed by adopting the Lenhartz method, or the plan recommended above. If chemical therapy is indicated, medicine such as the tincture of belladonna, from 5 to 15 minims (0.30 to 0.92 mil), may be introduced through the tube, directly into the duodenum, provided it is sufficiently diluted. If there is associated anemia, increasing doses of Fowler's solution, from 5 minims (0.30 mil), diluted with 15 mls (250 *m.*) of water, may be given through the tube two or three times a day. The writer has also used 5 grains (0.325 Gm.) of ichthyol, dissolved in 15 mls (0.5 oz.) of water, approximately a 2 per cent. solution, given through the tube two or three times a day in a case with associated ileocolitis, with apparently beneficial results. If the pancreatic ferments show diminished activity, substitution products may be likewise administered. The antacids are not, as a rule, indicated, and if they are, they should be administered by mouth to take effect in the stomach.

As stated before, the writer's experience with duodenal feeding for gastric or duodenal ulcer has been a limited one, and he, therefore, does not feel qualified to speak emphatically, either in praise or in criticism of this method. Nevertheless, his belief is that it is in no way superior to the method described above, and the suspicion may be tenable, that it is even productive of more harm than good, for the following reasons: The writer has seen several cases, well

tube-broken, who have become intensely intolerant to the presence of the duodenal tube when kept *in situ* for periods ranging from twenty-four hours to seven days. In all these patients the metal capsule was regurgitated back into the stomach, and this was preceded by midepigastric sensations varying from simple discomfort to acute pain. In two of these patients, occult blood, not present in the gastric contents or in the stools *before* treatment was begun, could be demonstrated after the tube was withdrawn. In one other case, the fluoroscopic study, which before treatment was begun corroborated the clinical diagnosis of duodenal ulcer only, on re-examination after the tube had been regurgitated, gave evidence strongly suggestive of prepyloric erosion, further substantiated by the fact that the patient had vomited small amounts of dark-brownish vomitus, strongly positive for occult blood, which led to the determination to withdraw the tube. The writer is of the opinion that in all of these cases the constant presence of the tube was the direct cause of these symptoms and findings, inasmuch as these patients progressed satisfactorily when placed upon the writer's usual plan of treatment. Again, it has been noted, frequently, that in fluoroscoping patients with the duodenal tube *in situ* there is a considerably increased peristalsis, with associated pylorospasm. Such an observation is not in accord with the writer's idea of gastric physiologic rest, and he believes that the passage of a bland liquid food over an ulcerated area will not cause as much irritation as the constant presence of a foreign body. Duodenal feeding may have a place in the treatment of ulcer, but the writer confesses to a doubt on this point.

#### TREATMENT OF SPECIAL SYMPTOMS AND COMPLICATIONS.

**Pain.** Pain is usually entirely controlled by complete rest in bed, by the use of external applications of heat or cold, and by the use of alkalies, silver nitrate, or belladonna; or, when due to hypersecretion, by aspiration of the stomach contents two to four times a day, using the duodenal catheter for this purpose, as described below. Where these measures do not suffice and epigastric pain continues to be a prominent symptom, it argues in favor of a mistake in diagnosis.

**Hypersecretion.** It is doubtful whether hypersecretion ever occurs in a simple ulcer, but it is one of the most frequent complications of ulcer, and is almost invariably due to pyloric obstruction, as a result of cicatricial contraction, adhesions, inflammatory edema, or pylorospasm. The last two lesions are amenable to medical management, while cicatricial stenosis will require surgical interference. The most effective relief is obtained by *periodically emptying the stomach of its irritating content*. This can be accomplished best by aspirating the stomach contents, and for this purpose the writer prefers to use a metal-tipped duodenal catheter, and to start emptying the stomach with a simple 1- or 2- ounce (30 or 60 mls) catheter-tipped syringe, after which the contents may continue to flow steadily from the stomach. If not, successive syringe-fuls may be withdrawn until the viscus is empty. It is good practice then gently to lavage the stomach by injection through the tube of a weak solution of silver nitrate, not more than 200 mls (6.67 ozs.) in a strength of from 1:6000 to 1:4000. This should be then aspirated and followed by 2 or 3 syringe-fuls of sterile water, and this by 200 mls (6.67 ozs.) of any bland alkaline solution. When the stomach is lavaged with such small amounts the tube should be withdrawn 3 or 4 inches (7.62 or 10.16 cm.), or until the metal tip lies just below the cardiac orifice of the stomach, so that the stomach-walls are gently sprayed from top to bottom with the lavaging fluid; to aspirate, the tube must be introduced further, so that the capsule lies along the greater curvature. This method is greatly superior to lavage by means of the usual stomach-tube, and is very well tolerated by most patients, especially when they have appreciated the amount of relief that can be obtained thereby. The frequency with which this should be done must depend upon the amount of the secretion and its acid concentration, but, as a rule, the empty morning stomach should be so treated, to prepare the patient for the day, and again at night to assure the patient a good night's rest. Three or four times in the twenty-four hours usually will be sufficient for the more pronounced cases.

In the writer's opinion this is a much more rational and effective procedure in combating the symptom than by

attempting to neutralize the acid secretion with alkaline medicaments.

In patients who are intolerant to the tube the throat may be sprayed with a 1 or 2 per cent. solution of cocaine, and after lavaging a 3 per cent. solution of anesthesin in 1 ounce of olive oil may be introduced through the tube.

**Vomiting.** Milder grades of vomiting can usually be relieved by the use of cerium oxalate, bismuth subcarbonate, and some form of magnesia. A combination powder, in small doses, may be made up to contain cerium oxalate, 3 grains (0.2 Gm.); bismuth subcarbonate, 10 grains (0.6 Gm.); magnesia usta, 5 grains (0.3 Gm.). Such a powder may be given four or five times at hourly intervals, and if relief is not obtained the vomiting is probably due to reflex causes. Of these, pylorospasm may be controlled by hypodermics of atropin in a dosage of  $\frac{1}{400}$  grain (0.0006 Gm.), to be repeated every second hour until physiologic effect is secured. One grain (0.06 Gm.) of powdered belladonna may be added to the foregoing combination, or belladonna may be administered in the form of the tincture, giving 10 minims (0.6 mil) every two or three hours, or until physiologic effects are secured. The use of these antispasmodics is likewise indicated where the vomiting is due to pylorospasm complicated by hypersecretion, but if relief from vomiting is not promptly afforded the writer has found nothing so effectual as *constant drainage of the stomach* for one or two hours by means of the duodenal catheter. This should be passed into the stomach, strapped to the cheek at the desired point, and with a small glass connecting tube a second piece of rubber tubing is attached, long enough to be carried down to an out-flow pail. The stomach contents should be aspirated by syringe and the stomach lavaged gently with syringefuls of sterile water, followed by mild alkaline solutions, such as soda bicarbonate 31 to 1000 mls (1.03 to 33.33 ozs.) of water, or a bland alkaline solution. The return of these lavaging fluids should be started by syringe or bulb aspiration, after which the stomach usually drains itself by gravity. Lavage should be repeated every fifteen minutes for one hour, using not more than 500 mls (19.99 ozs.) as the total for each lavage, and alternating the use of the alkaline solution with one contain-

ing 1 ounce (30 mils) of the tincture of belladonna in 500 mils (19.99 ozs.) of water. One or two hours of this treatment will usually be effective, but it may be continued for longer periods, if necessary. This is far easier for the patient than the exhaustion which follows repeated vomiting, especially if accompanied by much retching. Great care should be taken to see that the persistent vomiting is not due to an acutely *dilated stomach* due to a high duodenal obstruction, to angulation, or to a mesenteric ileus, as is occasionally seen in ulcer with complicating pyloric obstruction consequent to gastropsis. In such cases the foot of the bed should be well elevated, and the patient turned to the right or left lateral abdominal position, lying over a sand-bag or a bolster pillow, which is to be placed just below the level of the navel; the use of constant stomach drainage, as described above, should be continued, and, in addition to this, the writer in several cases has seen excellent results follow the use of the spondylo-therapeutic maneuver, described in detail on page 824, in the section on the treatment of acute dilatation of the stomach. In these extreme types of vomiting all food is to be withheld by mouth until the stomach becomes tolerant, when feeding is to be resumed most carefully, and with small amounts of liquids.

In cases where the vomiting is reflexly due to intestinal hyperperistalsis, it may often be relieved by the use of opium suppositories.

**Hemorrhage.** Adrenalin is the most efficient drug for the relief of this complication. It should be given in a dosage of 10 minims (0.6 mil) of the 1:1000 solution, and repeated every fifteen minutes for several doses. By its action the blood-vessel is contracted long enough for thrombosis to occur. Should this not prove effectual, lavage with ice-water has proved a safe procedure, being first recommended by Ewald, and since endorsed by many others. Before this is attempted it is wise to give a hypodermic of morphine to allay restlessness and to quiet the nervous apprehension of the patient, and the throat should be sprayed with a 1 or 2 per cent. solution of cocaine. It is important to perform lavage rapidly, and this can best be facilitated by the use of the Leube-Rosenthal method, with an irrigation tank connected



with the stomach-tube and attached to one limb of a glass connection, with an outflow tube attached to the other. This is a much quicker method, and easier for the patient. If restlessness continues no drug is of such service as morphin, which should be administered to the point of mild narcotization. If the hemorrhage be massive and the patient becomes exsanguinated, proctoclysis and hypodermoclysis should be practised. Direct transfusion may have to be resorted to in some cases, and the writer can thoroughly recommend the use of the Kimpton-Brown tubes for this purpose. Aside from the trick of properly coating the tubes with paraffin the technic of their method is extremely simple. It is wise to delay the building up of the body fluids by proctoclysis for perhaps an hour, to minimize the risk of dislodging a forming thrombus.

While profuse gastric hemorrhages are exceedingly terrifying to the patient and members of the family, and alarming even to the doctor, it is well to remember that few of such hemorrhages result in immediate fatality, and the greater majority of such patients may be saved.

An ice-bag should be immediately and constantly applied to the epigastrium. All food given by mouth should be stopped, and not begun again until all evidence of fresh melena has ceased, perhaps even until occult blood can no longer be found in the stools. These two measures prevent unnecessary movement of the stomach, and keep down gastric secretion, which might dislodge a thrombus or digest a clot. Nourishment is to be begun by rectal enemata. (*Cf.* p. 699). The patient is to be kept flat on the back, and not a single unnecessary movement allowed. When oral feeding is resumed it should follow the general plan outlined on page 703.

In a few cases surgical interference must be resorted to, and this is particularly indicated in the constant oozing from an eroded blood-vessel, which resists all medical measures, and in which the life of the patient is threatened, if the bleeding continues. The operative decision should be made promptly when it is seen that the hemoglobin estimations are constantly decreasing, and should not be delayed until the hemoglobin falls to a point that materially increases the operative risk. The stomach should be opened, the bleeding

vessel caught and ligated, the eroded surface cauterized, and a gastroenterostomy performed; in certain cases the ulcer may be excised. This sounds easy, but in reality it is no mean surgical feat, even in the hands of the most expert. The patient should be given transfusion with normal salt solution, together with proctoclysis and hypodermoclysis just before and again after operation.

**Perforation.** This complication is an imperative indication for an immediate laparotomy. The diagnosis should be easily made, and precious hours should not be wasted before moving the patient to the operating room and the septic scalpel. In probably no field of surgery does the life of the patient depend so much upon prompt and concerted action on the part of the physician and the surgeon, as the mortality materially improves in inverse proportion to the number of hours that have elapsed since perforation. In good surgical clinics most cases will recover if operated within ten hours after perforation. The prognosis is good even up to twenty hours, but after twenty-four hours the mortality rate rapidly increases. (*Cf.* p. 688.) It is still a mooted surgical question as to whether the perforation alone should be closed or whether a gastroenterostomy should be added. This is a matter for the surgeons themselves to decide, and doubtless hinges upon the surgical skill and manual dexterity of the individual operator.

**Secondary Carcinomatous Degeneration.** For this, prompt surgical interference is the one and only method of treatment. The burden of diagnostic proof should be laid upon every physician thoroughly to rule out the possibility of this serious complication. Every ulcer occurring on the gastric side of the pylorus should be under suspicion, and the case should be critically studied, both historically and by all laboratory maneuvers.

In any case undergoing a medical plan of treatment, in which there is a gradual diminution of hemoglobin, a persistent but insignificant loss of weight, the persistence of blood in the stools, and pain after one week of bed-rest and the use of heat or cold to the abdomen, an exploratory laparotomy should be strongly urged.

### CARCINOMA OF THE STOMACH.

Gastric cancer is a disease of the stomach in which a malignant neoplasm is implanted in the mucous wall or lining of the organ. It is usually insidious in its onset, and progressively fatal if unchecked. It is a disease of all ages, but occurs far most frequently during the fifth and sixth decades. It is always accompanied by disturbance of gastric function, and, while the symptoms may vary within wide limits, usually there is a progressive diminution of motor and secretory functions and deficient digestive activity, together with the development of abnormal chemical and bacterial findings. In advanced cases there are always associated constitutional symptoms of anemia, cachexia, and toxemia, with depressions of the nervous system. We are greatly indebted to Smithies<sup>18</sup> for the most recent exhaustive and statistical review of the subject of gastric cancer, and the writer cordially acknowledges the helpfulness of this source of information in compiling certain parts of this section.

Cancer, in general, is one of the problems of the day. If statistics can be relied upon the disease is on the increase, actually as well as relatively. Hoffman estimates that it is now 25 per cent. more common than ten years ago, and that this increase is true of the whole population of the world. Nor is this due alone to our diagnostic ability in its better recognition. In 1913 there were 75,000 deaths in the United States from cancer, of which gastric cancer furnished the highest mortality of all, and totaled 38 per cent. of all cancer deaths.

It is a hopeful sign, and somewhat indicative of the fact that the general practitioner is becoming more alert to the possibility of gastric cancer, that this disease is too frequently diagnosed without sufficient data. In support of this, Fenwick,<sup>19</sup> of London, states that of 56 cases admitted to his hospital service with a diagnosis of gastric cancer, 25 (44.7 per cent.) did not have it, yet this mistake occurred in illnesses averaging twelve months' observation, and Cabot<sup>20</sup> reports that of a large number of patients referred to him as gastric cancer there was 28 per cent. of diagnostic error.

It cannot be too strongly emphasized that there is *no*

cancer age. While cancer is more common in the years between 40 and 70, with the gastric incidence occurring in the sixth decade, nevertheless, cancer may, and frequently does, appear in the early period of life; and in Smithies' series of 921 cases, largely collected from the Mayo clinics, 10 cases were under 30, and 1 died at the age of 19. As regards race and nationality cancer is ubiquitous.

There are no occupations that really predispose to gastric cancer, but it seems to be more common among those who are more prosperous. In this regard Williams has noted that Ireland, with its rugged life and underfed people, has a much lower cancer death-rate than England, where prosperity is at a higher level, and similarly Hoffman,<sup>21</sup> in a study of life-insurance statistics, has shown that the mortality from cancer is much less among wage-earners than among well-to-do people.

There seems to be no particular kind of food-overindulgence which will predispose to cancer. Probably habitual overloading of the stomach, together with insufficient mastication, tends far more to the production of gastric cancer than does the type of food ingested. From Smithies' statistics, alcohol seems to play but a small part, whereas tobacco may be a more active etiologic factor, 15 per cent. of Smithies' cases being excessive smokers.

There does seem to be a proved connection between external abdominal trauma and the development of gastric-cancer symptoms, but it is more probable that the external injury may have caused an acceleration in a latent malignant process, rather than actually to have produced it. In addition to external abdominal injury, internal traumatic influences may be mechanical, chemical, biochemical, or parasitic, and evidence is accumulating that in certain cases each of these factors may predispose to gastric cancer. In regard to ingested parasites it is interesting to note the investigations of Febiger,<sup>22</sup> of Copenhagen, who found that certain laboratory rats were dying of cancer-like tumors of the stomach, and on examination he found that many of these tumors contained encysted nematodes. Febiger ascertained that these rats were largely secured from a certain sugar refinery that was infested with roaches. Examination of these roaches showed them to con-

tain worms. Febiger then collected large numbers of these roaches, and fed them to uninfected laboratory rats, and noted the development of cancer-like growths in the stomach of many of them. Commenting upon this, Smithies states: "While there is some doubt of the true carcinomatous nature of Febiger's rat tumors, the investigation is of value in showing the reaction of gastric mucosa to a parasitic irritant. It also shows the histologic difficulties of differentiating hyperplasia of a granulomatous type from the hyperplasia of true malignancy."

Heredity is a less important factor than we have been led to believe, occurring in only 5 per cent. of Smithies' 921 cases, although there are on record many well-known gastric-cancer families which have contributed the greatest amount of substantiation to our earlier beliefs. It would seem that there is a greater tendency of hereditary transmission of cancer of the uterus and mammary glands than of the type affecting the stomach.

Oral sepsis, undoubtedly, plays a large rôle in the establishment of gastric disorder, notably ulcer, and Smithies states that "gastric cancer and filthy mouths go hand in hand."

From the exceptional work of MacCarty and Wilson in the Mayo clinic we now know that chronic gastric ulcer is the most frequent disposing factor to gastric cancer, although many surgeons and clinicians affirm that the statistics of these workers are too extravagant in their association of carcinomatous degeneration with chronic calloused gastric ulcers, or the microscopic beginning of early gastric cancer in the immediate neighborhood of chronic ulcers.

The other gastric condition that may be a predisposing factor of cancer is the group of achylia, associated with the primary anemias, oral sepsis, intestinal autointoxication, and, less commonly, cholelithiasis. These achylia, which are regarded as benign, owing to their compatibility with years of good health, ultimately for no demonstrable reason become cancerous. Ewald, and particularly Wolff, of the Augusta Hospital in Berlin, personally emphasized to the writer the fact that they have watched certain benign achylia of several years' duration develop gradually into the malignant type, when checked by Wolff's soluble-albumin test,<sup>23</sup> and



ultimately confirmed by operation, and Wolff contended that such observations in the future would be more commonly recorded.

Gastric cancer is very frequently secondary to extragastric cancer, either by direct continuity of neighborhood viscera (liver, gall-bladder, pancreas and transverse colon), or by metastases by way of the lymph-channels from cancers of the breast, uterus or prostate.

Gastric cancer is a neoplasm having its microscopic beginning in atypical gastric epithelium, which shows a tendency to invade the surrounding tissues. Smithies has aptly stated that no one has ever histologically observed the *earliest* beginnings of any malignant process in human beings, nor has gastric cancer ever been experimentally produced in human beings. Therefore, we are left in ignorance regarding the early morbid histology. In many lower animals, and in many plants the experimental production of cancer has been successfully accomplished, and Smithies says that "it is now generally accepted that early malignant processes are in the nature of hyperplasia of already existing structural elements, . . . that a tissue reaction takes place which results in undifferentiated growths of a particular cell group, . . . and that commonly the specific function of the cells is lost."

Just what determines the beginnings of these cellular and intracellular deviations we do not know. They may be responses to irritants of various kinds, plus an inherent or acquired susceptibility of individual cells to be metabolically or histologically disturbed. We do know, however, that when the same irritant is experimentally introduced into different hosts, it frequently gives rise to cellular changes which may vary markedly.

The prognosis in any given cancer case depends largely upon the following points: the rate of abnormal proliferation of cell groups, the direction of their proliferation, the rate of their metastases, as represented by their accessibility to the lymph-channels; the effect such epithelial cells have upon the adjacent tubules in the production of retrograde changes; and complications, such as obstructions of the orifices of the stomach, contraction deformities of the stomach, hemorrhage and perforations.

Pathologically, carcinoma of the stomach may be divided into the following types: medullary, scirrhou, colloid and the *ulcus carcinomatosum*. While all of these types are fundamentally of epithelium derivation, yet the rate of their growth varies widely. Medullary and colloid cancers advance much more rapidly than the scirrhou variety, seemingly influenced somewhat by the state of nutrition of the individual, lean, spare, sallow patients appearing to tolerate cancer toxins better than do their more robust brothers. So, too, the location of the neoplasm governs its rate of growth, which is particularly rapid when it affects the cardia and the pylorus.

Cancer of the stomach is most likely to implicate the pylorus and upper curvature, and nearly all groups of statistics show about 60 per cent. of such a location. Cancers of the general stomach, of the posterior wall, the cardia, the greater curvature and the anterior wall, occur with decreasing frequency in the above order, while cancers of the fundus are least common of all.

Metastases control the prognosis, entirely irrespective of the size or rate of growth of the primary tumor in a given case, and the outlook is determined by the direction and extent of the lymph-gland invasion. The perigastric glands are commonly affected first, and next most frequently Virchow's gland, the left supraclavicular, this probably being due to the fact that the thoracic duct empties into the left subclavian vein at this point. Other important complications are those due to obstruction involving the pylorus or the cardia, malignant hour-glass constriction, hemorrhage and perforation.

**Diagnosis and Symptomatology.** In cancer of the stomach the most valuable diagnostic data may be obtained by the intelligent taking of the history, and the interpretation of these historical facts. It is not only the history of the present illness that concerns us, but a close inquiry into the presence or absence of symptoms of abnormal digestion that may have occurred for many years. We have learned that there is quite frequently a common sequence of events. Ideally the time to diagnose our gastric cancers is in their precancerous stage, and this can be most successfully carried out if we recognize that every gastric-ulcer history, especially if of long duration,

is potentially that of cancer. The symptomatology of this precancerous stage is essentially the same as that which commonly fulfills the accepted clinical requirements of gastric ulcer. There is a history of intermittent symptoms, with acute periodic exacerbations and equally sudden remissions of many weeks or months. The pain is of a boring, gnawing, sometimes burning, rarely colicky type (if associated with pylorospasm), and occurs one to two hours after eating, but the time varies according to the location of the ulcer and the amount and quality of the food. Furthermore, the pain is relieved by additional food-taking; or, by the administration of alkalies, and by lavage. Exacerbation of pain during the early-morning slumber hours is not infrequent. Vomiting is a common and most important symptom, and its type and time will vary according to the proximity of the ulcer to the gastric orifices, to the amount of cicatricial contraction, and to the amount of periulcerous inflammatory edema and congestion. This is the precancerous stage.

Carcinomatous degeneration around the edges and at the base of the ulcer may have taken place weeks and months before clinical symptoms of malignancy appear, and now the picture changes. The symptoms become continuous rather than intermittent; there may be no actual pain, but rather a sense of epigastric weight and discomfort. If pain be present it is usually constant; it is often of a dull-aching character, and shows much less tendency to food and medicinal relief; the appetite and nutrition, normally so well preserved in ulcer, may now begin to be affected; weight loss may be comparatively rapid, and is seemingly greater in the younger and more robust, and finally a degree of cachexia may be reached similar to that seen in the type of gastric cancer about to be described.

This type is the one that makes up the common textbook history of gastric cancer. It occurs usually at a later age period than that just described. In these patients we may have no history of pre-existing gastric disturbance. They tell us that until the onset of symptoms they never knew they possessed a stomach; that they could "digest wire-nails without distress," until suddenly, sometimes insidiously, they may become conscious of failure in appetite, with various aver-

sions to formerly well-liked foods, such as meats and sweets, and vague epigastric distress after meals may occur. At first this distress may be merely a sensation of epigastric pressure and heaviness followed by belching, with later sour regurgitations or water-brash. Pain may, or may not, be present, but it is usually of a dull-aching character, at first periodic and later continuous, depending largely upon the amount of food retention. Gradual progressive loss of weight and strength appears early, and its degree bears a relation to individual tolerance of cancer toxins on the one hand, and on the other hand to the situation of the cancer in relation to obstruction of the gastric orifices. Inasmuch as the commonest location affects the pylorus, obstructive symptoms appear relatively early, except in those rarer types of scirrhus cancer of the leather-bottle type, with rigid gastric walls and patent pylorus. When obstruction occurs vomiting becomes more common, and as the stomach dilates the vomiting gradually assumes the retention type. The vomitus may be odorless, but usually it is sour or rancid, and later becomes cheesy or putrid, according to the amount of cancer slough. It may be of any color, and if we wait for the classic coffee-ground type to appear, the case is usually inoperable. Diarrhea, of sudden onset and equally sudden cessation, may often be noted. Gradually the picture advances to that of profound cachexia with mental depression, often melancholia, with a palpable gastric tumor and enlargement of the superficial lymphatic glands. The condition is surgically hopeless.

It is not the writer's purpose to discuss in detail the possible information to be obtained from *physical examination*. In early primary malignancy there are no pathognomonic findings. In cancer associated with gastric ulcer the physical findings are those of the latter. When cancer is well established the diagnosis can almost be made from the foot of the bed by casual inspection. It might be well to mention the seven signs of inoperability of gastric cancers as tabulated by Smithies. They are:

1. Evidence of gland enlargement above the left clavicle.
2. Invasion of Blumer's rectal shelf.
3. Metastasis to the umbilicus.
4. Local or general increase in the size of the liver.

5. The presence of free peritoneal fluid.
6. Enlargement of inguinal lymph-nodes.
7. Palpable lymphatic metastases about the pylorus or along the lesser curvature.

In all cases, whether early or late, a thorough and complete physical examination is to be made, supplemented by detailed laboratory and röntgen-ray examination. No examination is complete without the use of the stomach-tube, and in few gastric conditions can so much important information be obtained. The empty stomach contents should be examined, and motor and secretory tests made. Every practitioner should train himself in carrying out these measures, should be able to interpret his results intelligently, and to properly weigh the diagnostic data. His office laboratory should be adequately equipped with reliable reagents to carry through most of the necessary tests, and sufficiently used to insure the saving in time by being familiar with technical methods. It is amazing the amount of laboratory work that even the busy average doctor can accomplish, if only he will do so. Daily practise makes perfect.

The methods of *diagnostic procedure* will vary somewhat in individual cases, but the following routine will answer for the most: The day before the patient's gastric analyses are to be made it is wise to have him take an ounce of castor oil at 3 or 4 o'clock in the afternoon. At 9 o'clock that night his dinner should be eaten, and may consist of any mixed meal containing meat, vegetables, particularly spinach or rice, and salad. At 10 o'clock about twenty seedless raisins should be eaten with their skins. The following morning, at a time varying from 7 to 9 A.M., a large-size stomach-tube should be passed, and the ten- or twelve- hour fasting stomach should be aspirated, the total amount recovered being measured and saved. By means of a 2- or 3- ounce (60 or 90 mls) glass syringe about 300 mls (10 ozs.) of warm water are introduced into the stomach, and reaspirated several times, so that the mucous membrane is gently flushed and the returning fluid contains small macroscopic flocculent particles. The total amount of fluid aspirated, which averages 150 mls (5 ozs.), is allowed to settle for a few moments in a conical vessel. The supernatant excess fluid is then poured off; a por-



tion of the sediment is pipetted off for a separate examination, and the remainder is added to an equal amount of 20 per cent. formalin. This sediment is then hardened by the usual methods, and paraffin-block sections are made and differentially stained.<sup>24</sup> This method of obtaining gastric sediments, with their subsequent microscopic study under all magnifications, may alone be sufficient to establish a positive diagnosis in many cancer cases. While the stomach is being rinsed by the above syringe method in the hands of an assistant, or even by the patient himself, auscultation and bimanual palpation of the gastric area and the left back can be quickly carried on in various positions, and the size, shape, position, and relative mobility of the stomach may be quickly ascertained. The tube is then withdrawn, and a secretory test-meal of 50 grams (1.607 oz.) of bread with 350 mls (11.066 ozs.) of water should be given and withdrawn fifty or sixty minutes later, by either the large or the small tube method. If time will permit, fractional analyses should preferably be made, and should always include fractional analyses for soluble albumin (Wolff-Junghan's reaction) to differentiate benign and malignant achylia.

All the materials extracted from the stomach should be measured, and the following points noted: the color, the odor, the relative proportion of residue and filtrate, the presence and amount of mucus, and macroscopic evidence of blood and bile.

Microscopic examinations should be carried out both in the unstained state and by the colored agar method, together with iodine and osmic acid preparation for the determination of food-rests. For its main objects the examination should determine the evidence of peptic digestion (digested protoplasm), the presence of cellular elements (epithelium, leucocytes, erythrocytes), hemin crystals, germinating yeast cells, sarcinae, Oppler-Boas bacilli, and other bacterial floras.

Food-rests from a twelve-hour fasting stomach, when macroscopic, are almost invariably indicative of pyloric obstruction, and to a less extent when found microscopically. It may be necessary, however, to repeat motor tests to be extracted at six- and eight- hour intervals, to detect lesser grades of motor insufficiency. The following chemical an-

analyses should be made: The amount of free and combined hydrochloric acid, the total acidity, the presence of associated organic acids, particularly lactic acid, and to a less extent butyric and acetic, and chemical tests for the determination of the activity of the gastric enzymes. The Gluzinski method of testing the gastric secretory response in the production of varying acidities to different test-meals is one of considerable aid in differentiating uncomplicated gastric ulcer from one undergoing carcinomatous degeneration.

While there are as yet no pathognomonic chemical or biologic tests for cancer, and while those thus far developed when positive only support the diagnosis of a cancer well established, nevertheless, it may be well to give a brief *résumé* of such of them as give the most reliable information. One of the best is the Wolff-Junghan reaction for the determination of soluble albumin, which is generally increased in cancer cases, either from albuminous absorption, from the presence of cancer-juice rich in albumin, or from the presence of a specific cancer-ferment which can carry proteid digestion to the final stage of soluble albumin. By this test the obtaining of 200 to 400, or more, units of soluble albumin is supportive of a cancer diagnosis. It is positive in about 80 per cent. of cases. The so-called "peptic index," by means of the Edestin test of Fuld and Levinson,<sup>25</sup> is sometimes helpful, inasmuch as it has been shown that cases of carcinoma with low free hydrochloric acid readings exhibit high peptolysis and low proteolysis, whereas benign peptic ulcers with low hydrochloric acid give low readings for both peptolysis and proteolysis. Again, the "formol index," suggested by Schryver and Singer<sup>26</sup> for the detection of specific ereptases in gastric juice, and modified by Sorenson and Schiff,<sup>27</sup> also is a good differential test, inasmuch as primary gastric cancer and ulcer carcinomatousum furnish a high formol index, averaging 20 to 22, whereas benign gastric and duodenal ulcers, benign achylia gastrica, and that associated with pernicious anemia give average readings from 10 to 14. The glycytryptophan test of Neubauer and Fischer,<sup>28</sup> and the modified tryptophan test of Weinstein,<sup>29</sup> are less important than those already mentioned.

Space does not permit a consideration of the examination

of the stool, except to emphasize the importance of determining the presence of altered blood by the use of the benzidine test for exclusion, checked by the guaiac test for provement. To be carried out properly the patient should be kept on a vegetarian diet for several days, and on a milk diet for twenty-four hours, and the second stool obtained after this should be tested.

While emphasizing the fact that such findings, unfortunately, rarely make for early diagnosis, the writer briefly summarizes the laboratory points most valuable in support of a diagnosis of gastric cancer:

1. Evidence of pyloric obstruction from the motor test-meals.

2. Evidence from secretory test-meals of a lowered hydrochloric acid output. (While this varies so greatly that one may expect to see all types of a normal or altered acidity, nevertheless, a lowered or absent free hydrochloric acid is suggestive, especially in the presence of a foreign acid, particularly lactic.)

3. The demonstration of Oppler-Boas bacilli in connection with positive or suspicious gastric sediment pictures.

4. A Wolff-Junghan reaction above 200 units.

5. A high formol index.

6. A high degree of peptolysis by means of the Edestin test.

7. The presence of occult blood in the gastric filtrate and stool extracts.

These singly or in combination are often more than sufficient evidence, but frequently they are obtained too late to be of much practical service to the patient.

The prognosis of gastric cancer depends mainly upon three factors:

1. How early the patient reports for medical examination.

2. On our ability to make an early diagnosis obtained from a searching anamnesis, careful physical examination, and the use of technical diagnostic tests.

3. Early operation and the ability of the surgeon mechanically to cope successfully with the conditions found on the operating table.

### TREATMENT.

The treatment of gastric cancer is entirely a surgical problem, except in such cases as prove inoperable. This is no field for the occasional surgeon. The writer believes in selecting a surgeon of long experience, with a sound surgical judgment of what *can* be done, what *ought* to be done, and what should *not* be attempted, a judgment that has been ripened by experience in daily operating, and particularly a surgeon who is interested in gastrointestinal problems, or in those of the upper abdominal zones.

**Surgical Treatment of Cancer.** There are five types of operations that are commonly done in the surgical treatment of gastric cancer. Taken in the order of their frequency they will be discussed as follows:

1. *Exploratory Laparotomy.* This operation should be heartily encouraged in cases suspected of gastric cancer when such have been properly studied from a historical, laboratory and röntgen-ray standpoint. Indeed, in many communities when advanced diagnosis is not obtainable, an early exploratory laparotomy best serves the interest of the patient. Where the possibility of cancer is concerned the writer unequivocally indorses exploratory laparotomy as a means to a final diagnosis, just as strongly as he condemns the too prevalent custom of exploratory laparotomy for the diagnosis of abdominal conditions that should be made non-surgically, if the patient is sufficiently and carefully studied, and in whom the stake of life or death is not unduly hazarded by the delay of a few weeks. But where cancer is a diagnostic presumption, exploratory laparotomy is thoroughly justifiable, and the earlier it is done the better, for by this means alone can the percentage of surgical cures be greatly improved, and the immediate or remote operative mortality lessened.

It is, as a rule, neither wise to tell the patient that gastric malignancy is suspected, nor to enter too closely with him into a differential discussion of his symptoms, inasmuch as apparent lack of certainty on the part of the diagnostician may cause a corresponding lack of confidence in the patient sufficient to cause him to postpone operative interference. While this is the rule, there are some patients before whom the facts

in their case may be fully outlined without shaking their belief that laparotomy is the wisest course, and without breaking down their courage and fighting spirit, should a cancer diagnosis be confirmed. In any event, some sensible member of the family or some reliable friend should be taken fully into confidence as to just what the diagnostic dilemma is, and why diagnosis by means of sight and touch in many cases is more reliable than the best of the laboratory maneuvers. In the hands of the capable surgeon of wide experience, often as well or better versed in gross and living pathology than is the laboratory expert, what the exploratory laparotomy discloses will determine the best operative procedure to follow. This will concern itself with one of the following operations or a combination of them.

2. *Resection of the Cancer-bearing Area.* This, the ideal operation, is often the most radical one, and, although it bears a higher immediate operative mortality, it insures the best and most enduring end-results in successful cases. In all ulcers on the gastric side of or at the pylorus, wide excision of the ulcer-bearing area offers the best preventive measure of gastric cancer, and the cure of incipient malignancy. In cases of gastric tumor of a carcinomatous type localized at or near the pylorus, with or without early implication of the perigastric glands, as wide a resection should be practised as is compatible with the surgical mechanics involved. Resection of one-third, or even one-half, of the stomach has been successfully carried out in a few cases, and either the continuity of the gastric lumen maintained by an end-to-end anastomosis, or a gastrojejunostomy performed for proper drainage. It is to be hoped that, as our diagnostic efficiency and our operative skill increases, a larger majority of our patients may be found in whom the radical cure for gastric cancer may be successfully attempted. However, the hope for this lies, to a great extent, in the hearty and sincere co-operation between the operating surgeon and the clinician, for it frequently happens that the latter, who has studied the case with painstaking care and minuteness, and in whom exploratory laparotomy discloses an indurated chronic ulcer, which no man, by sight or touch, can declare is or is not cancer in its incipient stage, is met with a disinclination on the part of



the surgeon to shoulder the increased operative responsibility of a radical resection. This attitude is to be strongly deprecated, and will be largely prevented as the operator gets farther away from the old standards of the "barber-surgeon," and studies each case with the closer scrutiny of the clinician. In such border-line cases as just cited, the field of rapid microscopic diagnosis, by means of frozen section studies, is just beginning to be practised outside of our larger surgical clinics, and, when capably carried out, may definitely settle such a diagnostic dispute.

The physician should make it a point to be present at all operations of patients he has studied, and if proper team-work is to be carried out he should be surgically clean, properly gowned and gloved, so that he can add the weight of his opinion, after personal sight and touch of the disease-bearing area, as to what surgical procedure should be attempted.

3. *Gastroenterostomy.* With or without partial resection, this is the commonest of all the palliative operations in the surgical treatment of gastric cancer. While not in any sense curative, it frequently prolongs life for many months, and even for a few years, during which time the patient enjoys marked symptomatic improvement. Indeed, in some instances the restoration to an appearance of health is so marked as to make one feel that a mistake in diagnosis may have been made, but sooner or later the advancing evidences of cachexia appear, and the subsequent march to the coffin is rapid. Gastroenterostomy is always indicated where motor obstruction affecting the pylorus is apparent. It is needless to say that the state of gastric motility by means of motor test-meals, supplemented by *x*-ray examinations, should be adequately determined before the exploratory laparotomy. While, theoretically, partial resection of a putrid, sloughing, inoperable gastric cancer may be indicated when combined with gastroenterostomy, on the grounds that it may partly arrest the absorption of toxins from this local source, nevertheless, it appears to the writer that in cases he has seen so treated much more metastatic invasion has taken place by way of the newly opened vascular and lymphatic channels, and it would seem wiser to get rid of such cancer toxins by means of frequent lavage with antiseptic solutions.

4. *Gastrostomy*. This palliative operation is indicated in cases in which the cancer invades the cardiac end of the stomach and prevents the passage of food by way of the gullet. By this means patients may be kept alive for several months, by direct feeding through the gastric fistula, although it often makes the patient's postoperative life more unbearable, and in the writer's opinion is against the principles of euthanasia.

5. *Jejunostomy* has been frequently performed as a substitute for gastroenterostomy in greatly debilitated patients, on account of the rapidity of the operative procedure and the minimal degree of subsequent surgical shock. It has, however, the same disadvantages that are associated with gastrostomy, and prolongs the patient's life for but a brief period.

**Medical Treatment of Cancer.** For those poor sufferers with inoperable cancer much may be done to make them more comfortable, although it may tax our resources to the utmost to accomplish such ends.

After an exploratory operation has been done, and the surgical verdict of inoperable cancer given, or when the attempt at radical resection has failed, and it becomes evident that there is local or metastatic recurrence, and when all palliative operative measures have been exhausted, the surgeon usually passes the after-treatment over to the physician, and is glad to wash his hands of further responsibility. To many a physician the medical handling of such incurable cases is often repugnant, many lose interest in the constant daily rehearsal of symptoms by the chronic and helpless invalid, together with the importuning of members of the family that *something* must be done. In such instances naturally it is often the sincere wish of the doctor that he might be allowed to hasten the coming of a speedy and painless death. But as long as we have our laws prohibiting the practice of euthanasia, and as long as our medical ethics are as they are, it becomes our manifest duty to prolong life to the utmost, and at the same time to make the voyage across the River Styx as easy and painless as possible. This requires patient persistence, tactfulness, cheeriness, and the constant instilling of courage to the very end. As already said, much may be done to alleviate unnecessary suffering, and the means

at our disposal may be grouped under four headings: general hygiene, mechanical, dietetic, and medicinal measures.

*Hygiene.* What has been said at length in the chapter on the treatment of ulcer in regard to hygiene, and the care of the mouth and its contents need not be recapitulated here, except to again emphasize the importance of these measures. (See p. 695 *et seq.*)

*Mechanical Measures.* Of all forms of treatment, nothing is more useful in the relief of symptoms than gastric lavage. Even in cases in which gastroenterostomy has been practised, lavage becomes imperatively indicated sooner or later.

The object of lavage in these cases is threefold: to remove from the stomach the accumulated food-products that occur in all cases of pyloric obstruction; to cleanse the mucous membrane, and, perhaps, to increase its secretory power (although in the majority of advanced cases we are dealing with a total achylia, due to permanent anatomical defects), and to get rid of broken-down cancerous *débris*, and thus prevent, as far as possible, the absorption of cancer toxins. To provide for the first object the best time to lavage is late in the afternoon, or, preferably, three or four hours after the evening meal, which will often insure the patient a better night's rest. To attain the other two ends, morning lavage of the twelve- or fifteen- hour fasting stomach serves better. In many cases it becomes necessary to practise lavage both morning and night. It is better to use a 32 or 34 F. calibrated stomach-tube, making use of the Leube-Rosenthal method by which lavage can be carried out much more rapidly and thoroughly. The lavaging solutions will depend upon the individual indications in any given case, and are described in detail in the chapter on Gastritis. (See p. 767.) To cleanse the stomach of mucus the alkaline solutions are best; to promote secretion a solution of sodium chlorid is probably most efficient, although silver nitrate or hydrochloric acid solutions may be used. Where there is considerable sloughing of a cancer mass with a foul, putrid odor of decomposing tissue, the writer prefers the use of a solution of potassium permanganate in a dilution strength of 1:15,000, gradually increasing to 1:10,000, which is to be promptly removed, and the stomach thoroughly rinsed with plain water. Other useful solutions

are 1 per cent. or 2 per cent. formalin, 1 per cent. salicylic acid, or resorcin 15 grains to the quart (1 Gm. to 1000 mls).

While the use of these antiseptic solutions may not accomplish much in themselves, nevertheless lavage uniformly results in symptomatic improvement, and the promotion of a sense of well-being. As a direct result patients eat better, sleep better and feel better, and the lavage should be continued until the weakness of the patient and the general exhaustion attendant upon it contraindicates its further use. At the conclusion of each lavage a small amount of liquor antisepticus alkalinus, or a solution of essence of peppermint, just pleasantly aromatic, may be left in the stomach on all occasions save when medicinal agents, such as castor oil, novocaine, cerium oxalate, and similar drugs are to be introduced through the tube.

In cancer affecting the cardiac portion of the stomach gastric lavage cannot be practised, but in cases in which the cardiac stenosis is not so great as to forbid feeding by the mouth, it is useful occasionally to wash out the esophagus, so as to prevent the occurrence of a localized esophagitis due to retained decomposing foods.

*Dietetic Measures.* In cancer of the stomach there can be no stereotyped diet which can be made to apply to all cases. The diet for each patient must be selected to meet the individual requirements, and the choice and character of foods will depend to a considerable extent upon the location of the cancerous growth. Naturally, cases in which there is pyloric growth sufficient to cause obstruction will have to be fed on a liquid or a soft mushy diet, in as highly concentrated a form as possible, and in amounts and in frequency to be determined by the emptying power of the individual stomach. In those cases in which cancer implicates the fundus, or the lesser curvature away from the pylorus, more latitude can be given in the selection of a semisolid or a solid diet.

It is very important that the likes and dislikes of the individual patient for food be catered to, and that they be allowed to eat as freely of such foods as they prefer, and which are adapted to the motor and secretory power of their stomach, without producing an aggravation of symptoms. Nothing reacts more quickly upon the general condition of

the patient than when he is obliged to follow an unpalatable, monotonous diet. With this plan there soon follows loss of appetite and suppression of whatever psychic juice may be present, due to mental rebellion.

Having settled upon the kinds of food that appeal to the individual patient, and which can be eaten with symptomatic agreement, the general principles of the food are that they shall be furnished in as bland a form, and in as fine a state of subdivisions as possible. Having placed the mouth and teeth in as good a state of cleanliness and repair as can be accomplished, careful mastication should be insisted upon. In other words, the stomach should be relieved of any unnecessary expenditure of digestive energy, both mechanical and chemical, which can be accomplished by other means (*e.g.*, proper preparation of food in the kitchen, thorough mastication, etc.). Of foods that are generally acceptable may be mentioned highly concentrated soups and broths of all kinds, boiled milk, milk foods, cream, buttermilk, a liberal use of butter and olive oil, minced chicken or other fowl, creamed oysters, soft-boiled or lightly poached eggs, and all kinds of non-scratchy vegetables, which are to be thoroughly cooked, passed through a sieve, and served in the form of a *purée*; zwiebach, dry toast, or stale bread may be eaten freely, if softened by dipping into soup or milk; meats, such as beefsteak, roast beef, lamb, and veal may be occasionally eaten, if they are first passed through a meat grinder. Should there be any marked pyloric obstruction, they need not be minced so finely, but may be chewed thoroughly, and the meat-juice swallowed, but the connective tissue and pulp discarded. Salads, if finely cut, may be eaten occasionally. Simple desserts, such as soft puddings, ice-cream, and the like, may be eaten, the preference being given to those made with milk or cream. Cooked cereals are permissible and useful when desired by the patient. Stewed fruits may be taken, provided that they do not increase gastric fermentation, and this applies also to other articles of diet. Non-aërated beverages may be used freely, and alcohol taken in medicinal amounts. It is well to avoid strong coffee, especially at night. As Ochsner suggests, it is well to avoid uncooked foods, particularly fruits, roots, and vegetables which are likely to be



contaminated by manure. Regarding the use of oils and fat, while they serve well to increase the caloric value of the diet, they leave the stomach slowly, and are prone to increase butyric acid fermentation. The quantity and quality of gastric secretion is a useful guide in the selection of individual food. (Cf. p. 692.)

In some cases where the pyloric obstruction is advanced it may be necessary to resort to duodenal feeding, if the catheter can be passed successfully. To this end the use of a duodenal tube without the metal tip, which can be passed over a silk thread, previously swallowed, serves the purpose best, inasmuch as the duodenal catheter can be withdrawn when not in use, stands less chance of food blockage while being used, can be more easily kept clean, and gives less discomfort to the patient. The foods suitable for duodenal feeding and the methods of their use have been discussed in detail in the discussion of gastric ulcer. (Cf. p. 703.)

*Rectal alimentation* may have to be used in such patients in whom sufficient nourishment cannot be provided otherwise. As a means of relieving thirst, the nutrient enema, preceded or followed by a short period of proctoclysis, is useful, and serves to combat acidosis. (Cf. p. 699.)

*Medicinal Measures.* Chemical therapy is indicated for the control of certain symptoms, but it should be supplemental to lavage. It is more important than dietetics in the treatment of gastric carcinoma, and drugs are useful for the correction of secretory defects, to promote better states of nutrition, and to control certain special symptoms, notably pain.

If there is hyperacidity or hypersecretion, the use of alkalis is indicated, and a wide selection of these agencies is available. Of most conspicuous importance are soda bicarbonate, various forms of magnesia, particularly if there is constipation, and the various alkaline waters. (Cf. p. 701.) In a certain number of cases, even though the gastric secretion is not high, symptomatic relief is obtainable from alkalies. As a rule, however, where gastric secretion is diminished or absent the use of the dilute hydrochloric acid in a dosage of 20 to 30 drops (1.25 to 1.9 mls), well diluted with water and taken in small amounts, with or after the meals, gives the better result. Acidol tablets or oxyntin (Fairchild's) may be

substituted. A good method of administering hydrochloric acid is in the form of acidulated milk. (See p. 778.) The use of the artificial enzymes is generally stated to be valueless. With a state of gastric anacidity the writer believes that an effective administration of enzymes can be accomplished from the use of pancreatin or pankreon in 50-grain (3 Gm.) doses, given in combination with an alkaline powder, two or three hours after eating.

As a stimulant to the appetite, aside from lavage, one may use various stomachic tonics, such as conduranago, nux vomica, hypophosphites and gentian. The writer has found the following combination of service in some cases:

- ℞ Tincture nucis vomici ..... ℥iv (15.0 Gms.).  
 Fluidextract condurango ..... ℥j (30.0 Gms.).  
 Tincture gentian. comp., q. s. ad ℥vj (180.0 Gms.).  
 Vel  
 Elixir hypophosphitum (N.F.)  
 q. s. ad ℥vj (180.0 Gms.).  
 M. S.: One or two teaspoonfuls (3.75 to 7.50 mls) to  
 be taken in a little water thrice daily before  
 meals.

To combat anemia one may substitute as a vehicle in the foregoing prescription the elixir of gentian and iron phosphate (N.F.), or the elixir of hypophosphite and iron (N.F.). Capsules of iron, quinine, strychnine and arsenic may be used for the same purpose, in the following combination:

- ℞ Ferri carbonatis ..... gr. iij (0.19 Gm.).  
 Quinini sulphatis ..... gr. j (0.06 Gm.).  
 Ext. nucis vomici ..... gr. ¼ (0.015 Gm.).  
 Acidi arsenosi ..... gr. ⅓₀ (0.0012 Gm.).  
 M. et ft. caps. no. j.  
 S.: Give such a capsule thrice daily.

As in so many other gastric conditions the writer thoroughly believes in the hypodermic use of drugs intended to stimulate the blood-forming organs, inasmuch as the dose given and absorbed can be more readily controlled without danger of upsetting the stomach. The contents of an ampoule containing 1, 2 or 3 grains (0.06, 0.13 or 0.19 Gm.) each of the citrate of iron and the cacodylate of soda, alone or in combination, may be injected intramuscularly in the buttocks once a day or every second day.

For the control of pain, when due to pylorospasm or to hyperperistalsis secondary to pyloric obstruction, the use of olive oil before meals, or a 2 to 5 per cent. solution of anesthesin in olive oil given before meals, or through the stomach-tube, following lavage, frequently proves effective, especially when combined with the use of alkalies. Authorities differ as to the use of orthoform, but in the writer's experience it has never proved as efficacious in cancer as it has in ulcer. Sooner or later, however, one will have to resort to various members of the narcotic group. It is wiser to delay their use as long as possible, and to begin with codein, giving  $\frac{1}{4}$  grain (0.16 Gm.) once or twice a day by mouth, and increasing the frequency as occasion demands. Later one must make use of morphine, which always should be given hypodermically and in a dosage and frequency only sufficient to control increased pain. Opium may be administered by bowel, if necessary, in the form of a suppository. Lockwood speaks highly of the use of the following prescription, which he believes is more easily tolerated than morphine:

R Pulvis opii denarcot. .... gr. ss (0.03 Gm.).  
 Pulvis aromatici ..... gr. ivss (0.28 Gm.).  
 M. et ft. caps. no. j.  
 S.: One capsule, two or more times a day.

Another useful formula for pain is the following, recommended by Bassler:

R Cocainæ hydrochloridum ..... gr. xj (0.7 Gm.).  
 Tinct. valerianæ ..... ℥ij (60.0 Gms.).  
 Aquæ chloroformi ..... q. s. ad ℥iv (120.0 Gms.).  
 M.  
 S.: Take 1 teaspoonful (3.75 mls) in water, through a tube, every four hours.

One-half to 1 grain (0.03 to 0.06 Gm.) of novocaine may be dissolved in 1 or 2 ounces (30 or 60 mls) of distilled water and introduced through the stomach-tube at the conclusion of the morning or evening lavage. The writer prefers novocaine to cocaine on account of its lower degree of toxicity.

While on general principles, it is much wiser to avoid the use of narcotics for the relief of pain, yet the writer believes that in gastric cancer, as well as in cancer of other organs,

we are dealing with an incurable malady, and if pain is obviously so severe as to need opiates, it is thoroughly justifiable that they should be not only used, but pushed, if necessary, to the complete control of unnecessary suffering. Cancer, as a rule, kills before the drug habit can be formed, and such patients are at least entitled to a painless death.

*Care of the Bowels.* The bowels should be kept well opened, preferably by the use of castor oil, which can be readily given through the stomach-tube at the conclusion of lavage, or administered in combination with malt-extract, beer, whisky, wine, or the syrup of sarsaparilla. No other laxative is as good in its effect upon gastric and intestinal fermentation. Calomel is useful on account of its antiseptic properties, and may be given once or twice a week in a single 5-grain (3 Gm.) dose at bedtime. It is better to use *liquid petrolatum* as an intestinal lubricant, in tablespoonful (15 mils) doses, once or twice a day, reinforced by cleansing enemata, and where laxatives have to be continued for a long time, in addition to those mentioned above, alophen pills and cascara sagrada have a distinct value.

For the control of other special symptoms the reader is referred to the discussion of Gastric Ulcer. (See p. 684.)

The use of the  $x$ -ray has long been advocated as a palliative, and even as a curative measure, in the treatment of inoperable cancers. Of its use in gastric cancer the writer knows nothing from personal observation, but he believes that it would prove far less successful than in the treatment of cancer involving the external organs or surfaces of the body. Certainly, treatment by  $x$ -ray should never be urged as an alternative to surgical exploration, whether early or late.

In comparatively recent years the use of certain metals, such as radium, mesothorium, selenium, and the like have found their way into the literature, and cases have been reported with good, bad and indifferent results from this sort of therapy. For some years to come the use of radium for this purpose will be greatly restricted on account of its scarcity and dearness, and its use can be made accessible only to the very rich. As a substitute radioactive waters have come more prominently to the fore, and when taken in sufficient quantities *may* prove useful in breaking down or taking

care of a superficial cancer slough, as in certain cases of colloid cancer.

In the seven or eight years that have elapsed since Hodenpyl brought forward the use of a specific serum therapy in the treatment of cancer, which ended with his untimely death, not much has been accomplished except to bring this form of treatment into considerable disrepute among the better members of the medical profession, who have lost faith in the different cancer autolysates, which have recently been urged on an unsuspecting public. On the other hand, the medical profession is awaiting with some interest further reports of the success of vaccination with split-proteids as inaugurated by the two Vaughans.

In a disease so deadly and dreaded as inoperable cancer we would eagerly like to grasp every therapeutic straw, on the ground that any form of treatment may be justifiable, yet it is well to remember that many of these later methods of treatment, even though they promise well, are still in their experimental infancy, and we should be conservative in our decision to adopt them blindly. Time alone will show their real utility.

### SARCOMA OF THE STOMACH.

Sarcoma of the stomach is a malignant neoplasm springing from atypical cell proliferation of connective-tissue origin, and, therefore, exists as a tumor primarily invading the walls of the stomach, with comparatively infrequent extension into the mucous lining or into the serous coat. Its course is similar, in many respects, to that of cancer, although its onset may be even more insidious and symptomless. Its duration is somewhat longer, as a rule, than that of cancer, but it invariably terminates fatally, and some cases run an exceedingly rapid course. Like sarcomas elsewhere, it metastasizes by way of the blood-vessels, and extragastric metastatic growths may be early and widespread. Metastatic sarcoma of the skin is not infrequent, and when present is a strong supportive evidence of the presumptive diagnosis. Unlike cancer, metastatic invasion is much less frequent than the spread of the process by direct continuity.

Sarcoma is a relatively rare malignant tumor of the



stomach. Since first described by Virchow there have been approximately more than 180 cases reported in the literature. Gossett,<sup>30</sup> in 1912, reported 171 cases that he had collected. Smithies,<sup>31</sup> in 1916, mentions 4 cases of gastric sarcoma observed among his 921 cases of gastric cancer, an incidence approximately of 0.25 per cent. Some writers, notably Fenwick,<sup>32</sup> feel that the true incidence of sarcoma among gastric neoplasms would be found to be considerably higher if accurate microscopic diagnoses were more carefully made in all gastric tumors, and he estimates its frequency at from 5 to 8 per cent. This view is shared by Perry and Shaw, who found 4 cases of gastric sarcoma in a series of 50 cases of gastric malignancy. These figures are doubtless too high, and probably Lockwood's,<sup>33</sup> estimate of 1 per cent. comes nearer the true facts.

Both sexes seem to be equally susceptible, although the age incidence shows it to occur in young adults far more frequently than cancer.

Our knowledge of the etiologic factors is even less than that of cancer. Brooks<sup>34</sup> reports a case occurring in the scar of an old bullet wound in the stomach-wall, which he ascribes directly as a result of local trauma. It is possible that in some instances the neoplasm in question occurs in the form of a sarcomatous degeneration of a benign tumor, such as a myofibroma.

Pathologically, sarcoma may occur as a localized tumor of, or as a diffused infiltration through, the wall of the stomach, and its size may vary from a small nodule to a tumor the size of a large grapefruit. Salomon<sup>35</sup> reports 1 case in which the tumor weighed fourteen pounds. It shows about equal tendency to extend its growth toward the mucous membrane and the serous coat, but microscopic examination of the glandularis has generally shown it to remain intact.

Histologically, sarcoma of the stomach may be classified in the same grouping used for extragastric sarcoma, although the round-cell and spindle-cell varieties are notably frequent when the stomach is the seat of this new growth.

The location of the tumor is more widespread than in cancer, although most frequently it affects either the pylorus or the greater curvature, but, even so, it rarely shows

the degree of pyloric obstruction so common in gastric carcinoma.

Gastric sarcoma may occur either as a primary or a secondary growth, although the latter form is exceedingly rare, and is likely to be of the melanotic variety. Of the different types, the round-cell sarcoma is more prone to undergo metastases, chiefly in the lymph-glands, and to a less extent in the liver and kidneys.

None of the symptoms are characteristic, and an accurate diagnosis cannot be made from the anamnesis. Cachexia and emaciation occur early. There may be few distinctly gastric symptoms. Even when the pylorus is involved, vomiting occurs much less frequently than in cancer, and is rarely of the retention type. Vomiting of blood, however, is rather common, and is usually more copious than is the rule in cancer.

A low grade of continuous fever has been noted in some instances. Anemia of the secondary type is usually marked, and is a rather suggestive finding, although it does not differ from that seen in severe chlorosis. The appetite is capricious, and early anorexia is not uncommon, although there do not appear to be the food aversions, which are not uncommon in cancer cases.

Pain is a rather constant symptom, and may vary from a sense of epigastric weight and pressure to true colicky, cramp-like pains, similar to those occurring in biliary colic; pain is a frequent complaint in sarcomas that progress in the direction of the mucous membrane.

Chemical analyses of the gastric juice, as a rule, yield little information. Secretory errors are less common than in cancer, although subacidities appear more frequent, notwithstanding the preservation of an apparently normal glandularis. Oppler-Boas bacilli and the presence of lactic acid have been noted.

The physical signs are typical only in the demonstration of an epigastric tumor which, by the tuning fork and auscultatory percussion, can usually be shown to be limited to the stomach. As a rule, the tumor mass is movable, and does not show the degree of fixation so common in cancer. Physical examination may give evidence of pyloric obstruction with secondary dilatation of the stomach. The spleen is occasion-

ally enlarged, and in lymphosarcoma Kundrat has noted that the lymphatic glands and commonly the lymphoid tissue of the tonsils are likely to be enlarged. The prognosis of gastric sarcoma is invariably bad, inasmuch as but few cases are diagnosed early enough to permit of surgical interference.

The treatment of sarcoma of the stomach is essentially surgical. In a suspected case the only hope lies in an early exploratory operation, with the possibility of finding the tumor sufficiently localized to justify a radical excision. In the presence of metastases resection is inadvisable on account of disseminating the pathologic process by way of the newly opened blood-vessels.

The non-surgical treatment of sarcoma is practically the same as that described for cancer, except that Coley's<sup>36</sup> mixed vaccine should be tried in all cases. If improvement does not follow within a few weeks, but little good can be expected from its further administration.

### GASTRITIS.

Gastritis is much too commonly diagnosed without sufficient pathologic evidence, and this is necessarily so in any disease in which it is difficult to furnish the pathologic proof. In all the field of gastroenterology no diagnosis is more frequently made than that of gastritis, and it is probably safe to say that in more than 50 per cent. of cases the diagnosis is wrong. Many cases of chronic appendicitis, and of chronic gall-bladder disease, giving rise to reflex gastric symptoms, are allowed to masquerade under the diagnosis of gastritis. It is very essential that the pathologic proof be furnished before such a diagnosis can be emphatically affirmed. There are two particular elements in the proof that should be demonstrated: first, an increase in intimately mixed, endogenous mucus to be seen in the gastric contents or vomitus; and, second, the demonstration of inflammatory elements in the gastric sediment, such as an increase of leucocytes, red blood-cells, and exfoliating epithelial cells of gastric origin. In the more fortunate cases one may recover bits of mucous membrane, sometimes including its whole depth from the periphery to the muscularis mucosa, and a study of this will

show pathologic alterations from which the diagnosis can be definitely made. As a general rule, pathologic defects which invade only the glandular portion of the mucosa indicate an acute process of the catarrhal type. Invasion of the deeper layers, with pathologic elements, means that the condition has become chronic; hence, with certain exceptions, we can divide our inflammatory diseases of the stomach into acute and chronic types, and both of these should be further classified into primary and secondary forms. There are four types of acute gastritis: acute simple gastritis, acute toxic gastritis, acute infectious gastritis, and acute phlegmonous gastritis. The differential diagnosis of these four types can best be built upon a platform of etiologic factors, rather than pathologic facts.

**Acute Simple Gastritis (Acute Catarrhal Gastritis).** The primary form, while less common, may result from mechanical irritants, such as coarse food, improperly cooked, and when eaten too rapidly and in too large amounts; from chemical irritants, such as the ptomaines in decomposing food, the excessive use of alcohol (an alcoholic debauch), the overindulgence in irritating condiments, or a mixture of rich foods that are not suitable to the individual's digestive apparatus. Among other chemical irritants which tend to produce a gastritis are the accidental ingestion of caustic alkalies or acids, or the long-continued medicinal use, in certain cases, of drugs such as arsenic, iron and phosphorus (the *gastrite medicamentouse* of the French writers). Thermal irritants, such as overindulgence in too hot or too cold foods or drinks, predispose to acute gastritis. Particularly if alcoholic, the iced drinks so common in America, are contributing factors of considerable importance. Secondary acute gastritis is a common complication of the acute infectious diseases, such as typhoid fever, pneumonia and influenza, and of such constitutional diseases as nephritis and gout.

Acute gastritis is common to all ages, and in the very young may be an extremely serious condition, and in the very old may result fatally. The prognosis, in simple acute gastritis, is uniformly good, except in those cases affecting the two age extremes. The symptoms usually promptly subside when the offending cause is removed.

The direct indications of treatment are the adoption of measures to combat an inflammation of the gastrointestinal tract, and, secondarily, to overcome such depressive symptoms as may occur as the result of a toxemia. At the earliest possible opportunity the stomach should be emptied of its irritating, often stagnating, contents, preferably by lavage, using 2 to 3 quarts (2 to 3 l.) of normal salt solution, or a decinormal solution of soda bicarbonate, which should be followed by some bland alkaline solution, such as the liquor antisepticus alkalinus. If the stomach-tube is not available, emesis should be secured by the administration of several glasses of warm water (to which may be added a teaspoonful of English mustard to the liter), or by inducing vomiting by tickling the pharynx with the finger. It is not wise to use irritating emetics. A single hypodermic injection of apomorphin,  $\frac{1}{10}$  grain (0.006 Gm.), may be used in cases in which emesis cannot otherwise be secured, provided that there are no symptoms of collapse. In such cases apomorphin should be given cautiously on account of its depressing action. Both Boas and Ewald recommend the use of ipecac and tartar emetic, in the following combination:

℞ Pulvic ipecacuanhæ ..... gr. xxij (1.5 Gm.).

Antimonii et potassii tartaris gr.  $\frac{5}{8}$  (0.05 Gm.)

Ft. chart no j.

S.: Take entire contents of powder either at a single dose, or in quarter amounts at intervals of ten minutes.

In children it is better to use the syrup of ipecac in a single dose of 1 teaspoonful (3.75 mils), or, in younger children, 15 or 20 minims (0.9 or 1.5 mils) every ten minutes until emesis has been secured. In all cases promptness in emptying the stomach is most desirable, inasmuch as it serves to get rid of the offending cause, and further protects the intestines against receiving the irritating material, either in whole or in part. The second important measure is promptly to empty the intestinal tract. Calomel is the drug *par excellence* for this purpose, on account of its antiseptic and disinfecting properties, in addition to its purgative effect. It is probably best to use two large doses, 5 or 6 grains (0.32 or 0.40 Gm.) each, as suggested by Ewald, taken one hour apart, than to use smaller amounts in divided doses. Before



waiting for the calomel to take effect the lower bowel should be emptied by a high colonic irrigation. The saline laxatives and castor oil had best be avoided. If the stomach is not retentive to the use of calomel, its second dose should be deferred until the second day after the attack. Should other than a physiologic diarrhea ensue, one of the best measures is to use a bolus of white clay or Fuller's earth in a dosage of  $\frac{1}{2}$  to 1 ounce (15 to 30 Gms.) suspended in a little milk or water, and this may be repeated in three or four hours, if necessary. After emptying the stomach no foods should be given by mouth for several days, in order to allow the inflammation of the gastric mucous membrane to subside, and also to suppress the secretion of gastric juices. Supportive rectal enemata may be used (see p. 699), and thirst may be controlled by sucking bits of cracked ice, and by the use of 2 or 3 liters (quarts) of normal salt solution or decinormal soda bicarbonate solution daily by proctoclysis, using the Murphy method, or any of its modifications. The mouth should be kept scrupulously clean by appropriate measures.

After two or three days of oral food rest, liquid diet may be instituted in the form of albumin-water, peptonized milk, strained oatmeal, or barley-gruel, and later thin broths, not made from meat-stock or extract. After from one to three days of liquid diet, depending upon the severity of the case, soft foods may be taken in the form of soft eggs, boiled or poached, oysters, oyster-broth, toast, bread and butter, custards, junkets, jellies, cereals, etc., and in a day or two more soft puréed vegetables may be added, and the usual diet may be resumed within from seven to ten days.

If *vomiting* should continue during the period of food-abstention the mechanical sedatives, such as bismuth subcarbonate or subnitrate and cerium oxalate may be used in the following combination:

R Cerii oxalatis ..... gr. xxv (1.6 Gms.).  
 Sodii bicarbonatis,  
 Bismuthi subcarbonatis ...ãã ðiiss (10 Gms.).  
 Div. in chartulas no. x.

S.: One powder to be taken in a little water every hour until relieved.

For the relief of *nausea* and milder forms of vomiting the sucking of cracked ice, to which has been added 1 or 2 teaspoonfuls of brandy, or *crème de menthe*, will often prove effective. If these measures do not suffice, the stomach had best be irrigated with a weak solution of soda bicarbonate, 1 or 2 drams to the quart (3.75 or 7.5 mls), and preferably by the small-tube-syringe method. (See p. 720.) For the control of *pain* it is rarely necessary, and much better not to use morphin. Symptomatic control can be secured, as a rule, by the use of hot or cold abdominal applications in the form of Priesnitz bandages, electric pads, or a mustard plaster, to be worn until the skin is thoroughly reddened, and then followed immediately by an ice-bag. If this does not suffice, codein sulphate in  $\frac{1}{6}$ - or  $\frac{1}{8}$ - grain (0.01 or 0.008 Gm.) doses may be given every third or fourth hour, or an opium suppository may be used, and repeated if necessary. If there are symptoms of prostration or collapse, the usual methods should be adopted, among which may be mentioned elevation of the foot of the bed, hot-water bottles to the feet, liberal use of blankets, proctoclysis, and the hypodermic use of strychnin or camphorated oil.

As a rule, *fever* is never very high, and it is well to avoid the use of antipyretics, on account of their depressing action. An ice-cap may, however, be worn. Following the attack it is wise to prescribe a tonic composed of strychnin or nux vomica and hydrochloric acid, in some stomachic vehicle, such as gentian. The following prescription may be recommended:

- ℞ Tinct. nucis vomicæ,  
 Ac. hydrochlorici dil. ....āā f3ij (7.5 mls).  
 Tinct. gentianæ comp...q. s. ad f3iij (90 mls).  
 M. S.: One teaspoonful (3.75 mls) in a wineglass-  
 ful of water before meals.

Acute catarrhal gastritis, secondary to the constitutional diseases, will rarely need other treatment than careful dieting.

**Toxic Gastritis.** The etiologic factor consists of the swallowing, by mistake or with suicidal intent, of acids, alkalies, metallic salts, and concentrated oils. Among these may be mentioned carbolic acid, oxalic acid, hydrocyanic acid, and the mineral acids, nitric, hydrochloric and sulphuric; caustic alkalies, such as lye and ammonia; metallic salts, such as

mercury, copper, silver, arsenic and phosphorus; raw alcohol, and various oils, like turpentine or copaiba. The pathologic lesion varies from simple hyperemia to ulceration, suppuration, with or without gangrene, and perforation. The character and the extent of the damage inflicted depends upon the amount of the poison taken, its character (corrosive or otherwise), its concentration, its length of stay in the stomach, and, to a great extent, upon the condition of the stomach, whether empty, partly empty, or full, and to a less extent upon the character of the food contained therein. Perforation is rare. Corrosive ulcers, discrete or confluent, are common. Maceration of the mucous membrane more commonly occurs after the use of caustic alkalies. A fatty degeneration of the glandulature is most extreme after the ingestion of arsenic and phosphorus.

The greatest amount of damage takes place at the points at which the poisons maintain the longest stay—the mouth and pharynx, the first and the terminal portions of the esophagus, the cardia, and the pylorus. As ulcers heal, cicatricial contracture may lead to stenoses and deformities. Esophageal, cardiac and pyloric obstruction are common sequels. The immediate prognosis naturally will depend upon the immediate damage inflicted locally, together with the toxic insult offered to distant organs, especially the kidneys and liver. If death does not occur, the prognosis will depend upon the character of the sequels.

Vomiting should be immediately induced by forcing the patient to drink several glassfuls of warm water, to which should be added the appropriate antidote. This is the first emergency measure. As soon as a stomach-tube can be obtained, the stomach should be washed out with warm water, medicated with the proper antidote. Lavage should be done early, to minimize the danger of perforation from too long continued maceration of the gastric membrane and wall. One should always risk the apparent danger of using the stomach-tube. Cases which will perforate will die, whether lavaged or not. Masterly decision is here worth infinitely more than watchful waiting. Apomorphin should not be used, as it simply adds to the existing depression. In an emergency case of poisoning it is often difficult to remember

the most appropriate antidote. In general, one may find ready to hand something that will prove effective if one bears in mind the general principles of chemical neutralization. For the mineral acids, nitric, hydrochloric and sulphuric, neutralize the acid with chalk, magnesia, washing soda, soapsuds, silver polish, whiting, or even plaster from the wall. These may be added to the lavage water, following which there should be introduced through the tube some demulcent, such as bland oils, olive or cotton-seed oil, milk, eggs, or mucilage of acacia. Alkaline carbonates are contraindicated, since they liberate carbon-dioxide gas, thereby distending the stomach and increasing the danger of perforation. Oxalic acid must be combated by only those alkalies which will form the insoluble and non-toxic calcium oxalate. Hence, one must select lime-water, chalk or wall-plaster. With both carbolic and phosphoric acids one should never use oil, because it increases the tendency of absorption of these two substances. For the neutralization of the alkalies, ammonia, caustic potash, caustic soda, or lye, one should use dilute vinegar, lemon-juice or orange-juice, and follow this with bland oils, milk, butter or lard.

If the patient does not die from the immediate toxic effects of the poison, the symptoms of pain, collapse, shock, suppression of urine, and other constitutional symptoms should be treated according to generally accepted principles. All foods should be withheld by mouth for a week or ten days, during which time rectal feedings should be carried out, together with the proper supportive measures. When oral feeding is resumed, it should follow the plan outlined for acute catarrhal gastritis, unless complicated by ulcer, when the course of treatment should differ in nowise from that of ulcer, as discussed on page 703. The sequels, esophageal, cardiac and pyloric obstruction should be treated according to the plans outlined in detail elsewhere. (See p 801 *et seq.*)

**Acute Infectious Gastritis.** Infectious gastritis is the name given to an acute inflammation of the stomach, in which the etiologic factor, or infecting agent, is a bacterium other than the true pyogenic group of staphylococcus and streptococcus. This term is likewise applied to those inflammations of the gastric mucous membrane resulting from the presence within

the stomach of vegetable and animal parasites. Where the infection is due to the Klebs-Löffler bacillus of diphtheria, it has been designated membranous gastritis (croupous or diphtheritic gastritis). It is of rare occurrence. Other infecting bacteria alleged to be potential factors of infectious gastritis are the typhoid, typhus and anthrax bacilli, and the pneumococcus and streptococcus, common to puerperal sepsis. Yeast, fungi, favus, maggots, roundworms and tapeworms have also been reported as etiologic factors. The pathologic lesion is very similar to that of a diphtheritic inflammation of other mucous membranes. The prognosis is grave, because the disease is usually secondary to a severe systemic infection, whose original focus is extra-gastric. Hence, the prognosis is that of the primary infection complicated by a severe gastritis. The symptoms are those of simple catarrhal gastritis, with the higher range of fever common to the primary disease. The treatment is partly expectant and partly specific. The management of the gastritis is essentially the same as that of acute catarrhal gastritis, with the addition to the lavaging fluid of germicidal antiseptics. (See p. 719.) In the future vaccines may have a more prominent therapeutic rôle than they have played in the past. Stock vaccines are in order, after proper identification of the infecting organism has been made, but should be discarded in favor of the autogenous as soon as the latter can be prepared. As a rule, this should not require longer than thirty-six to forty-eight hours after the specific bacterium has been isolated. Hence, not more than one or two injections of a stock vaccine will be required.

In bacteremic cases it is worth while trying the effect of direct transfusion of blood obtained from an individual who has been rapidly immunized by injections, in high dosage, of a vaccine made from the specific organism infecting the patient. The writer has seen one remarkable recovery from this method of procedure in a practically moribund case. The same care should be taken in the selection of the proper donor as applies to transfusions in general. The simplest method is the transference of the whole blood by the Lindeman method or by the Kimpton-Brown tubes. If transfusion cannot be carried out, the subcutaneous or intramuscular injection of the



immunized serum is the measure next of choice. The course and progress of the treatment should be governed by daily blood-cultures with colony counts. Among other measures calomel in small repeated doses is useful.

**Acute Phlegmonous Gastritis (Acute Suppurative Gastritis, Gastric Abscess).** This disease may occur as a primary lesion or secondary to acute bacterial infections, especially in the presence of a streptococcic bacteremia. As a primary disease it is extremely rare. Only slightly over a hundred cases have been reported since Borel first described it 160 years ago. The usual infecting agent in both the primary and the secondary form is the *Streptococcus brevis*, sometimes in association with bacilli of the colon group. A case has been reported in which the pneumococcus was regarded as the infecting organism.

All authors agree that the cause of the disease is invariably microbic. The writer has recently seen a case developing suddenly on the fourteenth day after a gastroenterostomy for an obstructive duodenal ulcer, and which terminated fatally on the fourth day after the onset of acute symptoms. In this case it may be worth while to include the principal facts in relation with this form of gastric disease.

At autopsy the site of the gastroenterostomy was operatively perfect, surgically clean, and showed no evidence of stitch abscesses or sloughing of tissues around the stitches. The entire stomach wall was very greatly thickened, especially so on the anterior wall from a point about the mid-fundic region to just above the pylorus. The serosal surface was shiny, had a waxy appearance, and was of a light grayish-pink color. The mucosa was smooth, shiny and edematous-looking; the rugæ were obliterated and the mucosal surface was of a deeper reddish pink, with a few minute points of a deeper red, suggesting petechiæ. The anterior wall, at the point of greatest thickness, which was midway between the greater and lesser curvature, about 3 inches (7.62 cm.) above the pylorus and at a distance of at least 1½ inches (3.81 cm.) above the level of the gastroenterostomy, was the seat of an intramural abscess. On cross-section the mucous membrane and the muscular coat with its attached serosa were greatly thickened, and the interstitial layers between contained a

necrotic slough 6 mm. ( $\frac{1}{4}$  in.) in diameter, and extending for a distance of 8 cm. (3.149 in.). The anterior wall, over this area, varied in thickness from 1 to  $1\frac{1}{2}$  cm. ( $\frac{3}{8}$  to  $\frac{5}{8}$  in.). On exerting pressure the softer points in this necrotic layer could be partly pressed out, and appeared like drops of very thick pus. Cultures made from the serosal surface recovered a Gram-negative coccus, and culturally not streptococcus, which was pathogenic to a guinea-pig twenty hours after inoculation. The same organism was recovered from the peritoneal cavity and the heart's blood of the guinea-pig. Cultures made from the mucosa recovered a second organism, a Gram-positive spore-bearing bacillus, which was not pathogenic to guinea-pigs. In serial sections, made from both the anterior and posterior walls, the bacteria could be demonstrated, and there was a marked occurrence of stratified thrombotic formations in widely dilated vessels. Otherwise the pathologic features were the complete cellular necrosis with the presence of polynuclear leucocytes, serum and fibrin, occurring between the muscularis mucosæ and the muscular coat. The glandular portion of the mucous membrane was strikingly well preserved, and the muscular coats were the seat of an early fatty degeneration. This patient was in good health, save for his ulcer symptoms, was a perfect surgical risk, and showed no evidence of oral sepsis or other bacterial foci of infection.

The *pathology* of this condition is very much as described in the foregoing paragraphs. The condition may occur, not only in the diffuse form described above, but in small, circumscribed, single or multiple abscesses. The *symptoms* are ushered in abruptly, usually with a chill, followed by a sudden rise in temperature to a rather high level, with acute upper abdominal pain, usually dry retching, and sometimes vomiting. Cases have been reported in which the vomitus contained pus from which the infecting organism has been recovered. Such diagnoses made during life are comparatively rare. There is usually a high leucocytosis. The physical findings are those of any acute surgical inflammatory disease affecting the peritoneum, with board-like rigidity of the upper recti muscles, and exquisite tenderness. In a time varying from one to several days, the patient goes into

sudden collapse with all the evidences of shock. This may be due to perforation, and is usually the beginning of the end, as the patient passes rapidly into a state of coma, soon followed by death. In the diffuse cases the lethal termination is usually prompt. In the circumscribed form the patient may live for several weeks. In the latter type, when diagnosed, prompt surgical interference offers the only hope of recovery. The mortality of unoperated cases is extremely high, something over 98 per cent. in the cases reported.

Aside from prompt surgical intervention, the *treatment* is purely symptomatic and expectant. The stomach may be lavaged with a solution of bichlorid of mercury in a strength of 1:10,000, or a solution of boric acid, using 1 ounce of the powder to a quart of water (30 Gms. to the liter). Probably these two are as good as any, although potassium permanganate may be tried in a dilution strength of 1:10,000. The supportive measures for collapse are the usual ones, proctoclysis, hypodermoclysis, heat, and the use of cardiac stimulants, such as strychnin, caffein and camphor.

**Chronic Gastritis (Catarrh of the Stomach).** This disease may occur in either a primary or secondary form, according to the etiologic factors involved. In the primary form the pathologic lesions of the gastric mucous membrane may be caused from the ingestion of substances that are irritating to the mucous membrane. These substances may be of either a mechanical character, such as the coarser, 'scratchy' forms of food, improperly cooked, and usually eaten too hastily without proper mastication, and in too large amounts.

Thermal irritants, such as foods that are too hot or too cold, or foods that are too highly seasoned may produce a chronic inflammatory state of the stomach, if their use is too long persisted in.

Among chemical agents the long-continued use of alcohol stands first in importance and incidence, and forms a distinct type of chronic gastritis which can be differentiated from the others. The café, grill, or bar-room habitué is particularly prone to this condition, especially when indulging in late suppers or dinners selected from the usual café menu. Among other chemical irritants the medicinal use of iron and arsenic, and to a less extent silver and phosphorus, may terminate in

chronic gastritis if long continued, and especially in individuals with a sensitive gastric mucosa.

In its secondary form chronic gastritis commonly accompanies all cases of chronic passive congestion of the splanchnic vessels, and is habitually seen in portal obstruction, in cirrhosis of the liver, and in valvular and muscular lesions of the heart when a state of incompetency has been reached. Gastritis likewise frequently accompanies other organic diseases of the stomach, such as cancer and ulcer, and especially those cases complicated by pyloric obstruction. Again, it is practically always associated, sooner or later, with constitutional diseases of long standing, prominent among which may be mentioned nephritis, tuberculosis, syphilis, diabetes mellitus, pernicious anemia, Banti's disease, and Addison's disease.

It is somewhat of a paradox that, with all the etiologic factors so commonly encountered, one may yet affirm that gastritis is too frequently diagnosed on general principles, rather than on the furnishing of pathologic proof. Nevertheless, this is true, and no such diagnosis can be a sound one unless it is made upon the examination of one or more specimens of gastric contents obtained direct from the stomach, in both the fasting and digestive periods. The direct pathologic evidence can frequently be demonstrated by the study of gastric sediments obtained from the fasting morning stomach, and by this means two distinct forms of gastritis may be classified on a pathologic basis.

**Hypertrophic Glandular Gastritis.** This presents the clinical features of an acid gastritis, and here the pathologic diagnosis depends upon the findings of fragments or flakes of gastric mucous membrane which show a well-marked hyperplasia of the glandular elements, and in which the individual cells retain good staining power. This applies most particularly to the base or fundic portion of the glands, whereas the cells toward the periphery show granular protoplasmic degenerations, loss of staining power, and absence of nuclei. This peripheral portion frequently desquamates or sloughs off, and is found in isolated areas in the microscopic field. The interglandular stroma is infiltrated with an increased number of leucocytes; lymphocytes predominate in the more chronic process, while the polynuclear varieties, if in abundance, will



indicate either an acute gastritis or an acute exacerbation of a chronic process, provided that ulcer and cancer are excluded. Fragments of recovered mucous membrane may show enlarged or dilated venules, and areas of pigmentation and congestion may be seen. This is particularly true of those cases in which chronic passive congestion of the splanchnic vessels is an etiologic factor. The amount of mucus is usually increased, although this is not as invariable a finding as in the pathologic process next to be described.

**Atrophic Gastritis.** This type presents the clinical features of a subacid or anacid gastritis, and here recoverable bits of gastric mucosa show a considerable diminution in the number of gastric tubules, and a marked irregularity in their distribution; their alignment is very imperfect, and few glands can be traced from fundus to neck. The cells stain poorly, and show mucoid degenerations of the protoplasm with marked vacuolization and a notable absence of cell nuclei. Frequently gastric cells are seen lying in the lumen of the tubule, separating or completely broken away from the basement membrane. Indeed, all of the epithelium may be completely denuded from the tubule, leaving simply an empty space in the mucosa, bounded by a skeleton framework representing the basement membrane. The leucocytic infiltration is usually of the lymphocytic type, and areas of venous congestion are relatively infrequent. In the more chronic stages there is an increase of the interstitial connective-tissue elements, occurring between the tubules. The quantity of mucus is almost invariably increased, and may be found as a deep layer of mucus lightly attached to the peripheral portion of the fragment of mucosa, or as islands of mucus occurring in isolated portions of the section.

In the same microscopic field, or in other portions of the sediment in the same case, may be found practically normal glandular elements, and clinically such cases usually show normal gastric secretion. This may furnish a plank in the argumentative platform that the findings of such microscopic fragments of the mucosa, showing various pathologic states, may not represent a true picture of the amount of organic damage, or the degree of functional power of the stomach as a whole. Nevertheless, there is a very close parallelism



between such pathologic evidence obtained by sediment study and the clinical features in the given case.

From a clinical standpoint, chronic gastritis may be classified into three forms:

1. Gastritis with normal or increased hydrochloric acid and enzyme output (gastritis *acida*).

2. Gastritis with a diminished hydrochloric acid secretion, but in which the ferments may be either normal or moderately reduced (gastritis *subacida*).

3. Gastritis with total failure of secretion of hydrochloric acid and the ferments reduced (gastritis *anacida*) or absent (*achylia gastrica*). Where the ferments are absent it is of extreme practical importance to demonstrate the presence or absence of the proferments. Their continued absence indicates very little likelihood of restoring the gastric secretion by any form of treatment.

The course and prognosis will naturally depend upon two factors: first, the efficiency with which the exciting cause is not only removed, but prevented from recurring; and, second, the amount of pathologic damage inflicted before this can be accomplished. Much depends upon how deeply the inflammatory lesions have penetrated the gastric mucosa. In those affecting the more peripheral portion the outlook is much more favorable, inasmuch as the glandular elements of all secreting membranes have a tendency to regenerate themselves from below, the dead or functionless cells being desquamated from the surface. Not only is the pathologic lesion concerned in the depth of its penetration at any given point, but, also with its lateral extent throughout the entire secreting surface. Naturally, one expects that the hypertrophic glandular type (gastritis *acida*) would be much the more likely to recover, after the exciting cause has been removed. Such is, however, not always the rule, but where acid symptoms are continuous, notwithstanding appropriate treatment, one will do well to suspect a chronic ulcer or a state of vagotony. In those cases with normal amounts of gastric secretion the situation is usually more simple and the prognosis better. Where the gastric secretion is very greatly diminished or absent, especially the latter, and supplemented by the sediment picture of an atrophic gastritis, one can, as a rule, hold out little hope

of restoring the secreting functions of the stomach to any point that will aid in digestion, and the aim of treatment should be directed toward protecting the intestines and conserving their function. If this is accomplished the patient will continue in good digestive health, notwithstanding the absence of gastric secretion.

### TREATMENT.

The treatment can be divided into *prophylactic* and *direct*. The teeth should be put into good order, false teeth being provided if necessary, and thorough mastication should be insisted upon, not alone that the food may be properly brought to a state of fine division, but to stimulate the flow of saliva, and thus to secure the benefits obtained from salivary digestion.

The habits of the patient should be critically interrogated and a proper hygiene ordered—fresh air, home hydrotherapy, and a proper balance of rest and exercise. In short, anything that tends toward building up the general level of health will bring about the betterment of the local condition, insure the stomach a better blood-supply, and aid in the regenerative repair of the pathologic process. Also, in the way of direct removal of causative factors, all irregularities in diet should be corrected, both as to the kind of food and the manner in which it is eaten. For the business man or woman hasty lunch-counter eating should be forbidden; iced drinks or very hot soups interdicted; mixed alcoholic beverages, especially in the concentrated forms, forbidden, except that in certain cases a little whisky, well diluted in a glassful of water, may be taken with or after the meal. This is particularly necessary in the chronic gastritis due to alcoholism in which, in exceptional cases, it is neither safe nor possible to stop the use of the drug abruptly. Attendance at dinners, public or social, where rich viands in many courses are served, should be avoided; the use of tobacco restricted, especially in the hyperacid form of gastritis; and the chewing of tobacco emphatically forbidden.

If iron, arsenic, and the like; or the habitual use of laxatives have been contributing etiologic factors, they should be promptly discarded, and other measures adopted in their place.

The *direct treatment* may be divided into dietetics, mechanical, balneological, electrical and medicinal.

**Dietetics.** The general principles of the diet will vary according to the clinical type of gastritis. Hence, the need of the proper classification of these cases after determining the state of the gastric functions, both secretory and motor. If secretion is increased, both in amount and concentration, the diet plan elaborated in the treatment of hyperchlorhydria (see p. 764) should be followed, and need not be repeated here. In general, a mixed diet should be given, the chief essential being that the foods are bland and non-irritating, both chemically and mechanically, and are furnished in a state of fine subdivision, so that less effort will be needed on the part of the stomach to grind them to a chyme suitable for entrance into the intestines.

For the first few days it is probably best to place the patient on a mixed liquid and soft diet, in order to give the stomach a partial rest. The number of meals, and the amount of food eaten at each, of course, depends upon whether atony is present or not. In this event, five or six small meals, with a restriction of fluid, is preferable to three meals of the customary size. In uncomplicated gastritis the motor power of the stomach usually is not affected, with two exceptions, the hypomotility associated with a markedly excessive secretion of mucus, and the hypermotility usually accompanying atrophic gastritis or the achylic states. In the cases in which hydrochloric acid and the ferments are increased salivary digestion will be inhibited, and farinaceous and carbohydrate foods will be less well borne than the proteids. Where the hydrochloric acid and the ferments are reduced, foods rich in the native proteins or dense with connective tissue will be difficult of digestion, and only the softer proteins, such as fish, eggs and milk, should be used, and the diet built up in cereals, breadstuffs and vegetables. When the secretion of hydrochloric acid and the ferments is totally absent, the use of proteins must be greatly reduced or entirely discarded, and the patient placed very largely upon a vegetarian diet. In this group of patients, too, it may be permissible to use some of the predigested protein foods, such as somatose and laibose, which are mentioned merely as examples.

In all of these three forms the caloric value of the diet may be built up to any point desired by the liberal use of fats in the form of butter, cream, olive oil and cheese.

Where constipation is present buttermilk is often helpful; likewise honey, cooked fruits, or the use of the "fruit formula," with or without senna, prepared as follows:

Take 6 apples, 6 pears, 12 dates, 12 figs, and 12 prunes.

Chop and cut up finely and place the pulp and juice in a saucepan with 1 quart (liter) of water, using senna. Tie up the senna-leaves in a piece of cheesecloth, and place them in the saucepan with the fruit mixture, and boil mixture slowly down to 1 pint.

Strain through gauze. Bottle and keep well corked in a cool place (preferably the ice-box).

For use take 1 tablespoonful followed by a glass of cold water in the morning on rising and at night before retiring.

Salt should be used freely in subacid and anacid gastritis, since it has been shown to stimulate the secretion of gastric juice, and, conversely, its use should therefore be reduced in the treatment of hyperacid forms. Similarly, such stimulating articles as salted and smoked fish, herring, mackerel, anchovy, caviar, and the sharp condiments, which are definitely contra-indicated in hyperacid gastritis may find a place in the dietary of the subacid and anacid forms.

The following diet is suggested as illustrative of the general principles stated above, and is particularly the diet of choice where the *gastric secretions are normal or diminished*:

On arising, a cup of hot beef-tea made from any good meat extract or a cup of hot water with  $\frac{1}{2}$  a teaspoonful (1.875 mils) of table salt in it. This has a tendency to promote gastric secretion, and should, therefore, be limited to those cases with diminished secretion.

*Breakfast.* An orange or a grapefruit, or their juice. A saucerful of some well-cooked cereal, such as farina, or cream of wheat may be eaten with cream and sugar. One or 2 eggs, lightly boiled, poached or scrambled, and some crisp breakfast bacon, discarding the dense connective-tissue portions; or a small portion of salted or smoked herring, or mackerel, in markedly subacid cases. Stale bread, zwiebach, or dry toast, with a liberal amount of butter, may be eaten freely. Brown,

graham, or whole-wheat bread should be selected if the patient is constipated, and honey or marmalade may be added. Buttermilk, if constipated; otherwise, a cup of cocoa with cream and sugar, or a glass of milk. Coffee had best be entirely avoided, although a cup of weak tea is permissible if especially desired.

*Luncheon.* A soup, preferably clam or meat broth, although cream purées, such as tomato, potato, asparagus and pea, are permissible. Creamed chicken, lamb-hash; good, tender ham, cut fine and thoroughly masticated; lamb-chop, creamed fish, or oysters in any form except fried. A thoroughly cooked mashed or baked potato. One vegetable selected from the list outlined for dinner. The choice of any light dessert, such as milk, rice, tapioca, junket, cornstarch, blanc mange. Ice-cream, ices, and iced drinks are not allowed. As a beverage a glass of milk or buttermilk, or a glass of one of the medicated mineral waters, discussed later, and to be selected according to the state of the gastric secretion. Stale bread, zwiebach, or dry toast with butter. All foods may be well seasoned, and simple relishes may be eaten.

*Dinner.* A soup as at luncheon. A small piece of steak or roast beef, chicken, or broiled or boiled white fish with butter sauce. It is a good rule that if meat is eaten at one meal, fish should be eaten at the other, and the use of meats should be somewhat restricted, in proportion to the lessened gastric secretion. Mashed, baked, or creamed potatoes. The choice of any two of the following vegetables: Creamed or boiled cauliflower, spaghetti or macaroni, well-cooked rice to be eaten with butter, spinach, squash, asparagus, tender string beans. Any vegetables that can be put through a colander and puréed with cream are permissible. A simple salad with French dressing. The same choice of desserts as at luncheon. The same choice of beverages with the additional choice of a little whisky, well-diluted with water, or a glass of good claret or Rhine-wine. Champagne and all aerated wines, and all malt beverages, such as beer and ale, are distinctly injurious and should be avoided. Foods may be well seasoned, and simple relishes eaten.

If a gain in weight is desired, midmeal feedings may be selected from the following:



Six or 8 ounces of milk with 1 raw egg well-beaten in, to which nutmeg may be added. This may be still further richened by adding one-third cream, and may be flavored with vanilla or chocolate, or any fruit juice if there is a distaste for milk. Buttermilk, kefir, koumiss, or matzum may be chosen instead. A few salted crackers, liberally buttered, should be eaten.

These midmeal feedings, likewise, apply to such cases of gastritis as are complicated by atony, remembering however, that in such instances the use of liquids with meals is to be avoided.

In cases of anacidity the proteid content of the above menu must be greatly reduced or omitted. The proper dietary to be followed in anacidity will be found in the discussion on Dietetics. (See p. 764.)

**Mechanical Treatment.** Lavage stands first and foremost as an efficient agent in the mechanical treatment of uncomplicated gastritis, but its use should be limited, with one exception, to those cases which show an overabundant secretion of mucus that is intimately mixed with and surrounds the food particles in a very tenacious mass, and thus not only prevents the ready admixture of food with the gastric juices, but likewise delays the exit of the chyme. The one exception is in those cases of hyperacid gastritis in which the secretion of mucus is low, and in which the mucous membrane is subjectively sensitive to the burning and corroding action of the gastric juice. In such instances the mucus is protective, and its secretion should be encouraged by lavaging with stimulating medicated solutions such as silver nitrate in a dilution of 1:3000 to 1:1000.

Lavage in all cases should be practised for a *short period* only, and if definitely satisfactory results cannot be accomplished in three or four weeks' time, its further exhibition should be discontinued. Lavage serves to cleanse the stomach and rid it of mucus, which is often thick and ropy, and plugs up the lumen of the secreting tubules; it further stimulates the secreting power of the oxyntic and enzymotic cells, and, in addition, in neurotic patients frequently creates a sense of general well-being in a purely psychic manner. The best results from lavage are seen in those cases of gastritis with

diminished secretion and increased mucus. In anacidity and dry achylia less can be accomplished, since the pathologic process is more extensive, although patients aver that they feel greatly refreshed after this internal toilet. Where atony is a complicating factor, and there is some degree of stagnation and fermentation, lavage is doubly indicated, but should be practised with caution. Not more than 400 mils (13.34 ozs.) of the lavaging fluid should be introduced into the stomach at any one time, and care should be taken that the amount recovered equals that introduced. In gastritis with atony the best results are obtained where electrical treatments are combined with lavage. (See p. 773.) The selection of the proper time for lavage is important. By far the best time, in most cases, is to wash the stomach in the fasting state, before breakfast. This serves to cleanse the stomach of its accumulated mucus, and to prepare it for the day's work. It is a time, however, that is more practical for hospital cases than for those who are to be treated in the office, the more convenient time for the latter being about the noonday period, or from 5 to 6 o'clock in the evening, which usually means one hour before the next meal and four or five hours after the last one. In gastritis lavage need never be practised oftener than once a day, and this for never more than the first week, gradually reducing the number of treatments to every other day, then every third day for a total period of not more than three or four weeks. The temperature of the lavaging fluid should be from 100° to 105° F. (37.8° to 40.4° C.), where a cleansing effect is desired. In the treatment of an associated atony, alternate hot and cold douches, by means of the double tank (Leube-Rosenthal method), tend to improve the motor defects. The amount of lavaging fluid introduced at any one time should not exceed 500 mils (16.67 ozs.) before a similar amount is recovered, and in atony, as above stated, 400 mils (13.34 ozs.) should be the highest limit. As to the selection of the lavaging fluid, the choice is a wide one. Numerous medicated solutions have been recommended from time to time, but aside from the few that will be discussed, the writer has seen no advantageous results from their use which could not be achieved by normal salt solution or plain water alone.

In chronic gastritis with increased mucus, lavage with an alkaline solution is by far the best, inasmuch as this serves

to liquefy and dislodge the tenaciously adherent mucus from the gastric mucosa.

The best alkaline solution is soda bicarbonate in a strength of 1 tablespoonful to each quart (16 Gms. to each 1000 mls), or lime-water, 1 ounce to each quart (32 Gms. to each 1000 mls). After lavaging with this, a solution of the tincture of hydrastis, 1 tablespoonful to each quart (16 Gms. to each 1000 mls), or fluidextract of hydrastis, 1 to 2 teaspoonfuls to each quart (4 to 8 Gms. to each 1000 mls), seems to act favorably as an astringent, and to a great extent prevents further secretion of mucus. This is particularly helpful in gastromyorrhea. In gastritis with marked subacidity one may use pure hydrochloric acid, 1 teaspoonful to 1 quart (4 mls to 1000 mls), which is stimulating to the glandularis, and is also an excellent antiseptic. This solution, however, should not be used except in the later weeks of treatment when the excess of mucus secretion has been diminished. In chronic gastritis of the advanced achylic types, even in the presence of an excess of mucus, lavage seldom does good, and may be harmful. This does not apply, however, to the psychic achylas. In hyposecretion, associated with hypomotility, when not organic or malignant, lavage for short periods with pure hydrochloric acid, as above, or with a 1:1000 silver nitrate solution serves to stimulate the glandularis. As a bland alkaline solution the writer prefers the liquor antisepticus alkalinus, and makes a practise of terminating lavage in hyperacid cases by introducing, through the stomach-tube, 1 ounce (30 mls) of this solution, diluted with an equal quantity of water, and leaving it *in situ*.

In hypersecretion lavage is harmful with the following three exceptions:

1. In hypersecretion or hyperacidity where there is little or no mucus present, lavage with a silver nitrate solution, as stated above, will give marked subjective relief by acting as an irritating stimulant to the glandularis, by increasing the amount of mucus secretion, which serves as a protectant from the irritating gastric juice. This subjective relief continues, even though the chemical analyses show a higher acidity in the end than was present in the beginning, provided that the mucus secretion has been relatively raised.

2. In gastritis with hypersecretion, when complicated by fermentation, lavage will prove of benefit, and in such cases the writer prefers to use a weak solution of potassium permanganate in a strength of 1:10,000 to 1:15,000.

3. In the irritative form of gastritis (hypertrophic glandular gastritis, gastritis acidæ) a short exhibition of the alkaline lavage solutions above-mentioned often secures good symptomatic results.

In lavaging all cases of gastritis in the fasting state the total amount of lavaging fluid needed will seldom exceed 2 quarts (2000 mls). This amount of lavage can be easily carried out by means of the Leube-Rosenthal irrigating method in from three to five minutes, depending upon the tonicity of the gastric muscles, which largely determines the rate of the return-flow. Where solutions, such as silver nitrate, potassium permanganate, or pure hydrochloric acid are used, they should be followed by a gastric douche with plain water. At the termination of lavage various mechanical sedatives to the stomach, such as cerium oxalate, bismuth subcarbonate, or syrup of acacia, may be introduced through the tube. Likewise, this is an excellent and easy method of administering castor oil, should its use be indicated.

The direct treatment of the gastric mucosa by means of gastric sprays, powder insufflations, and the like, by means of specially constructed apparatus has not proven particularly beneficial in the writer's experience. It is almost needless to state that the proper dietetic and hygienic management of the patient is quite as important as the local treatment of the stomach.

**Balneological Treatment.** The use of medicated mineral waters is of considerable value in assisting the treatment of the various forms of chronic gastritis, although in the writer's opinion this is by no means one of the therapeutic essentials. These mineral waters, according to their type, serve either to stimulate the gastric glandulature toward an increased secretion, or partly to neutralize the overproduction of acids. With this object in view, the particular water prescribed for each case should be intelligently selected. Unfortunately, it is true that this particular field of natural therapy has not been sufficiently studied in its connection with the various

springs in this country. When this has been systematically undertaken it is quite likely that some of our natural waters will rival or excel in their effects those of the most exploited European spas. This is a most timely necessity, inasmuch as the present European holocaust will, for some years to come, affect the popularity and accessibility of most of the foreign water-cures.

While the medicinal value of these waters is considered a therapeutic entity by European writers, and occupies a large space in their literature, the use of the imported waters, or of their desiccated salts, has not proven brilliantly efficacious in the writer's experience, and it is doubtless a fact that the beneficial results obtained by residence at such foreign spas comes from the freedom from business and social cares, from the outdoor exercise, from the enforced regularity in habits of eating, drinking and sleeping, from the abstinence in alcoholic beverages, interdicted at such places, and from the constant flushing of the kidneys and skin that naturally would follow drinking of large amounts of these waters, rather than from any essential indispensable medicinal value of the waters themselves.

As a rule, motor defects contraindicate the use of mineral waters, especially those of the aërated type. Particularly is this important in the motor defect due to atony, and patients thus affected should be cautioned not to drink more than one glass at a time, and this at a midmeal period, or at least, not within two hours after the last meal. Those patients who are of a nervous disposition, or who are greatly run down should not be given purgative waters, or at least, never in amounts sufficient to cause watery movements. Where there are no motor errors, and secretory defects alone have to be considered, the selection of the water to be used may be determined by the following facts:

In anacid or achylic states the saline waters which contain chiefly sodium chloride and carbon dioxide are indicated. The best of the European waters for this purpose are those of the Rokoczy Spring at Kissingen. The salts from this spring have been carefully studied, and the artificial substitute, the Sal Kissingense Factitium, has been introduced into our newer formulary with a dosage of 1 gram to a glassful of



water (15 grs. to 8 ozs.). Other European springs which enjoy a good reputation for the treatment of these conditions are Homburg (the Elizabeth Quelle Spring), Wiesbaden (Kochbrunner Spring) and Ems. In this country the Congress Spring at Saratoga has very much the same chemical composition as that of the Kissingen water. The action of these waters is said to dissolve gastric mucus, to augment the secretion of hydrochloric acid, and to improve the appetite. Experimental work upon dogs in the laboratories of Pawlow and others has shown that the gastric secretion, after the introduction of these saline waters, has been from 50 to 75 per cent. greater than with that obtained with ordinary water. In psychic achylia reports have been published of a re-establishment of hydrochloric acid after a sojourn at Kissingen or at Saratoga Springs.

The water should be given cold in atonic constipation, warm if there is spastic constipation, and hot if there is diarrhea. Not more than one glassful should be given, and on an empty stomach, about one hour before the meal. The best results are obtained in those cases of subacid gastric catarrh where hydrochloric acid is still present, although in diminished amount, and where the mucus secretion is increased.

For patients in whom the gastric secretion is high, with an overproduction of mucus, the alkaline mineral waters are to be selected. Among these the following may be recommended: Carlsbad water, which, in addition to being alkaline, has a laxative action, and is, therefore, useful in cases with constipation. When this is present the waters should be taken cold. The natural water from the spring has a temperature of 144.8° F. (63° C.), and the hotter it is taken the less effect it seems to have upon the bowels, and especially is this true in atonic constipation. Where the original Carlsbad water cannot be secured, the desiccated salts, prepared and dispensed by Eisner and Mendelsohn, may be substituted, or our pharmacopœial preparation, the *sal carolinum factitium*, may be used. In this country the water of the Bedford Springs is very similar in its chemical composition to that of Carlsbad. Both contain sodium sulphate, sodium chlorid, sodium carbonate, and free carbon dioxid. None of these laxative waters should be given in sufficient amount to cause diarrhea.

As a general rule, one glassful is to be taken hot, on an empty stomach, one-half hour before breakfast, and should this not cause diarrhea, half a glass may be taken before luncheon and before dinner. Pluto (French Lick Springs) and Mount Clemens waters in this country, and the Hunyadi Janos and Apenta waters abroad, are somewhat too drastic in their purgative laxative effect to be serviceable in the treatment of hyperacid gastritis.

The Carlsbad treatment, given hot and in small doses, is indicated in hyperacid gastritis with constipation, in chronic gastric and duodenal ulcers, and in chronic ileocolitis. It should never be given in functional gastrointestinal diseases, even though it may be otherwise indicated, where the etiologic factor can be traced to a disturbance of the nervous system. It invariably makes nervous patients worse.

In those patients in whom constipation is not a feature the best waters to combat the acidity are Celestins Vichy (or our official substitute, the *sal vichianum factitium*), Victoria Brunnen, or, in this country, the water from the Hathorn Spring at Saratoga. These waters all contain sodium bicarbonate and carbon dioxid. Where there is an associated atony the waters containing carbon dioxid are contraindicated. Where anemia is present, Levico, Mild, or Schwalbacher waters may be used.

**Electrical Therapy.** The use of electricity during the past five years has come rapidly to the fore in the treatment of gastrointestinal diseases, and in chronic gastritis, as a supplement to dietetics and lavage, it has proved a valuable addition to our therapeutic armamentarium.

There is still considerable controversy between physiologists and clinicians as to what we should ascribe the clinical improvement following the use of electricity, the favorable evidence of which is rapidly accumulating. The physiologists have tenaciously held the opinion that the glandulature and musculature of the stomach will not respond to an electrical current, and that the beneficial results clinically observed must be due to psychic effects alone. With this view the writer emphatically disagrees. With an intragastric electrode *in situ* and an extragastric electrode placed externally at any point of the body, but preferably over the third to the eighth

thoracic vertebra, and the current turned on, it can be easily demonstrated clinically that the current is completed by observing the gastric contractions produced, which can be readily palpated through the abdominal wall, and which can often be subjectively felt by the patient. Furthermore, it has been demonstrated, by means of fluoroscopic examinations on patients being so electrically treated, that there is not only a rhythmical contraction of the viscus, but an increase of the peristaltic waves.

These treatments may be given both intragastrically and by the percutaneous route, but the former is very much the more efficient. During the earlier days of its use there was some uncertainty as to which types of current should be used to overcome the different secretory and motor defects, but the trend of opinion now is that the faradic current is useful for retoning gastric musculature, with or without atonic ectasia, where the pathologic defect is not so great as to prevent respiration of muscle tone. This current, likewise, stimulates an increase of gastric secretion. It is useful, too, in cases of abnormal relaxation of the pylorus and the cardia. The galvanic current seems to act particularly well as a sedative for abnormal disturbances of gastric sensation, and is particularly efficacious in the treatment of gastralgias and hyperesthesias of nervous origin. It is generally conceded to be the current of choice in cases of hyperacidity or hypersecretion. In nervous patients, with secretory or motor, rather than sensory defects, the faradic current exercises a psychic effect that is often not accomplished by the galvanic. More recently the sinusoidal current has been developed and perfected, and, in the writer's opinion, is the current of choice in the treatment of atonic stomachs, inasmuch as it guards against overfatigue of the gastric muscle on account of the long period of relaxation between contractions.

For the treatment of subacid gastritis, after the inflammatory phenomena have been controlled, and the diminished secretion alone remains, or in cases of delayed secretion, as diagnosed by fractional gastric analyses, the writer has had some brilliant results with the use of the Bergonie apparatus modified by Naegleschmidt. This device delivers an interrupted current of the sinusoidal type, which can be made as

slow or fast as desired. It is especially useful where there is an associated atony or an atonic ectasia, provided the pathologic defect is not too extreme (paralytic atony). The intragastric method gives considerably quicker results, although satisfactory improvement will follow the extragastric route. With the Bassler electrode the ordinary patient does not object to, and often prefers, the intragastric method. It can be easily passed with a little initial swallowing assistance on the part of the patient, and is quite comfortable when once beyond the glottis. In subacid cases the positive pole is attached to the intragastric electrode, as it causes less electrolysis, and the extragastric electrode, in the form of a hand sponge, is attached to the positive pole. This electrode may be either a large one, 6 inches (15.24 cm.) long by 3 inches (8 cm.) wide, which can be strapped in position over the vertebral region from the third to the eighth thoracic vertebra, from which point the sympathetic nerve-supply to the stomach emerges from the spinal cord, or a smaller sponge electrode may be carried down the sides of the neck and over the sternomastoid muscle, to stimulate the pneumogastric nerve lying beneath it. Where there is an associated relaxation of the abdominal musculature the external electrode may be placed over the abdomen for a few minutes of each *séance*, to secure abdominal contractions. Where the intragastric method is employed the duration of each treatment should not exceed ten minutes, although longer sessions may be given by the percutaneous route, and the writer prefers to interrupt the treatment every two minutes and give an interval of one minute rest. Where the sinusoidal or galvanic currents are used intragastrically the strength of the current should not exceed 15 to 20 milliampères, and the faradic current should be well within the limits of tolerance of the patient. Treatments may be given every day for a week, every other day for a week, and then at intervals of twice or once a week, dependent upon the progress of the case. In gastric atony, to secure satisfactory results in the way of permanency, the treatments should be continued for two or three months. They should always be given on an empty stomach, preferably before breakfast, or three or four hours after the last meal. In the latter case it is better to lavage



the stomach, after which 200 to 400 mls (6.67 to 13.34 ozs.) of water, or, preferably, normal salt solution, may be left in the stomach to serve as a conductor for the electricity and to prevent burning of the mucous membrane from contact with the electrode. Should lavage not be performed, the patient should drink one or two glasses of water, which will serve a similar purpose.

**Medicinal Treatment.** When the dietetic and physical methods of treating the various forms of gastritis have been properly carried out, there usually remains but little indication for the use of chemical therapy, and there is no doubt that in any case the use of the methods outlined above are far more beneficial than the *indiscriminate use of drugs*. Especially is this true, as is commonly the case, when various chemical agents are prescribed *before* a knowledge of the gastric secretory and motor state of the patient has been learned. Careful and complete gastric analyses should be made in every patient, and test-meals should be used to determine motor as well as secretory defects. Furthermore, in many cases *fractional* gastric analyses will be necessary to properly interpret the question of disordered secretion. In any form of gastritis with an associated motor defect in which the stomach does not fully empty itself during the time interval between meals, and the stomach still contains a miscellaneous chyme, tenaciously bound together by mucus, and with mucus densely adherent to the gastric mucosa, it is unreasonable to believe that *any* medicinal agent designed to take effect *upon* the gastric mucous membrane, or to be absorbed *through* the gastric mucous membrane, can possibly do so when confronted by this mechanical impediment.

Many badly disordered stomachs are made worse by the indiscriminate or irrational use of chemical agents. The first requisite is that the stomach should be cleansed of its mucus before medicinal agents can be made effective. When this has been accomplished, and when gastric chemistry has been determined, there are certain drugs which may be prescribed, alone or in combination, according to the indications peculiar to the individual case.

Where there is hypersecretion or hyperacidity the use of antacid powders may be given after meals, just before the



highest point of secretion has been reached. Bicarbonate of soda forms the important foundation for all such powders. If there be an associated constipation the light oxid of magnesia may be added as in the following prescription:

R Magnesia ustæ ..... gr. x (0.6 Gm.).  
Sodii bicarbonatis ..... gr. xx (1.2 Gms.).

Ft. chart. no. j.

S.: Suspend the powder in 1 ounce (30 mls) of water, and take after meals and at the time directed.

In patients with gastric hyperesthesia or gastralgia the use of the mechanical sedatives, such as bismuth and cerium oxalate, may be employed. The following prescription can be recommended:

R Cerium oxalatis ..... gr. v (0.3 Gm.).  
Bismuthi subcarbonatis ..... gr. x (0.6 Gm.).  
Soda bicarbonatis ..... gr. xx (1.2 Gms.).

Ft. chart. no. j.

S.: Suspend the powder in 1 ounce (30 mls) of water, and take after meals and at the time directed.

Where bismuth is indicated as a mechanical sedative and hyperacidity is not present, bismuth subnitrate is better than bismuth subcarbonate, and should be given before meals rather than after meals, and in somewhat large doses. It is often good practice to give a single dose of 30 to 50 grains (1.8 to 3.0 Gms.) suspended in half a glassful of water on a fasting stomach before breakfast. This will often control gastric hyperesthesia for the day. In cases with hypersecretion the use of silver nitrate by means of the nine-day cycle, as recommended by Lockwood (see page 708), is often beneficial, provided that the hypersecretion is of a functional type. As already stated, where the mucous secretion is low and the gastric acidity is high, the use of a silver nitrate lavage in a dilution strength of 1:3000 to 1:1000 is a useful procedure, or the tincture of belladonna may be given before meals, starting with 5 drops and increasing a drop each dose, but keeping well within the physiologic tolerance of the patient. This should not be continued for too long a period.

In gastritis with diminished or absent secretion the use of hydrochloric acid is logically indicated.

Where the hydrochloric acid is merely diminished the dilute hydrochloric acid may be given in a dosage starting with 10 drops (0.6 mil) and increasing 1 drop (0.06 mil) each time the medicine is taken until 25-drop (1.5 mls) doses have been reached, and thereafter maintaining this maximum. It should be given well diluted in 4 to 8 ounces (120 to 240 mls) of water, one-third to be taken at the beginning of the meal, one-third at the close of the meal, and the balance one-half hour later. It should be taken through a glass tube. If there is diminished appetite one may add to the foregoing 1 teaspoonful (3.75 mls) of a stomachic, of which the writer prefers either the compound tincture of gentian or the fluid-extract of condurango. The administration of hydrochloric acid, in addition to its effect as a partial aid in gastric digestion, fulfils an important purpose in stimulating the secretion of pancreatic juice.

Another way of administering hydrochloric acid is by means of acidulated milk, which can be prepared by adding to a glassful of milk sufficient dilute hydrochloric acid as to cause a reaction for free acid to Congo paper. In constipated cases, due to hepatic torpor, not only may the gastric secretion be stimulated, but also the flow of bile increased by the use of the dilute nitrohydrochloric acid, in a dosage of from 3 to 5 minims, well diluted and taken through a glass tube after the meal. For this purpose, and as a tonic, for those cases of gastritis secondary to a prolonged acute infection, the following prescription of Hare's is an excellent one:

**R** Acidi nitrohydrochlorici

dilutæ ..... fʒj vel. fʒij (4-8 mls).

Tincture nucis vomicæ... fʒj (4 mls).

Tinct. cardamomi comp.: fʒij (60 mls).

Tincture gentianæ

compositæ .....q. s. ad fʒiv (120 mls).

**M. S.:** One teaspoonful (4 mls) is to be taken well diluted with water after meals.

Aside from its effect as a gastric stimulant and as an excitant of the hormone secretion, it is a generally accepted fact that the administration of dilute hydrochloric acid in the dosage commonly employed is entirely ineffectual as an active

agent in gastric digestion. In this regard it has been stated (Sippy) that it requires approximately 100 drops of dilute hydrochloric acid to aid in the digestion of 15 grams of albumin. Therefore, with a patient on a diet calling for 100 grams of protein, it would require 600 or 700 drops of the dilute hydrochloric acid. This amount cannot be administered except by means of the stomach-tube.

In antacid gastritis, where the administration of acids is not well borne, they should be withdrawn and the gastric state kept alkaline with bicarbonate of soda or other antacids, the diet being arranged upon a plan suitable for intestinal digestion, which sometimes may be aided by the use of taka-diastase, pancreatin (preferably pancreon), or inspissated bile-salts in a dosage of 5 to 10 grains (0.6 to 1.2 Gms.), given after meals. There are various preparations, such as oxyntin, acidol tablets, gastrinin, and similar well-advertised preparations which have no especial advantage over the official dilute tincture of hydrochloric acid, besides being considerably more expensive.

### ALCOHOLIC GASTRITIS.

This type of gastric catarrh, if of long standing, usually is accompanied by marked subacidity or anacidity, with an overproduction of mucus, and where cirrhotic changes in the liver have occurred, achylia is commonly encountered. Properly treated, a symptomatic cure can be accomplished, but a real or anatomic cure will depend upon the amount of structural damage already inflicted, and upon the reparative power of the individual mucous membrane. The essentials of treatment consist of the immediate total withdrawal of alcohol, the use of chloral and bromides administered by bowel to allay restlessness, and the application of detoxication methods by flushing the intestines, kidneys and skin. To this end one may use hot applications to the abdomen, and later hot packs or vapor baths, the free use of some good spring-water to act as a diuretic, and the use of small doses of the compound jalap powder to aid in the withdrawal of fluid from the tissues. By these means much of the alcoholic bloat can be removed, and later on the atonic muscles may be restored by electrical or hydropathic methods, assisted by voluntary exer-

cise. It is needless to state that the patient must be a "blue ribboner" to the end of his days, if anything like a satisfactory result is to be secured. During the first part of the active treatment a liquid diet should be maintained, which later may be increased to a point compatible with the patient's gastric chemistry.

Where the stimulating effect of alcohol is suddenly withdrawn the cardiac symptoms will often require digitalis, which may be given alone or combined with capsicum, the latter being extremely serviceable in alcoholic gastritis. The following prescription is useful:

℞ Tinct. capsici ..... f3j (4 mls).  
 Tinct. digitalis ..... f3ij (8 mls).  
 Tinct. gentiani comp...q. s. ad f3iij (100 mls).  
 M. S.: Take 1 teaspoonful (3.75 mls) in 1 or 2  
 ounces (30 or 60 mls) of water before meals.

An ice-bag should be kept over the precordia, and the use of depressants should be avoided. To allay restlessness in cases verging upon delirium tremens, in addition to the use of chloral and bromids, Lockwood speaks well of the effects of paraldehyd, given two or three times a day in teaspoonful (3.75 mls) doses, although it has a disagreeable taste and odor, and is likely to upset the stomach.

After the acute symptoms have subsided the further treatment does not differ in anywise from that of other forms of chronic gastritis, except that alcohol must be completely interdicted.

### SYPHILIS OF THE STOMACH.

This is a disease of the stomach caused by the *Treponema pallidum* of Schaudinn, which may affect the mucosa and sub-mucosa of the gastric wall, either alone or in combination. It may result from both hereditary and acquired syphilis, but in both types it should be considered a tertiary lesion. While it represents one of the rarer implantations of visceral syphilis, it may yet prove to be far more common than we were once led to believe. About twenty-five years ago Chiari<sup>37</sup> reported 243 post-mortems on syphilitic individuals, and found definite syphilitic involvement of the stomach in but two instances, an incidence of 0.8 per cent. More recently Stolper

has autopsied 86 patients who have died of syphilis, and has found 2 cases showing definite syphilitic invasion of the stomach. Averaging this total of 329 cases with four instances of gastric syphilis, the frequency of this disease, from a post-mortem standpoint, is 1.2 per cent.

It is to be presumed that this estimate will prove far too low, in view of the comparatively large number of cases of gastric syphilis that have been published in recent years. For instance, William Gerry Morgan<sup>38</sup> reports 8 cases occurring in his practice in the past twelve years, the majority of which occurred within the last three years, which were diagnosed partly from historic and symptomatic evidence, and partly by *x*-ray, serologic and operative means. None of these cases came to autopsy. It is manifestly a difficult matter accurately to determine its true frequency. Theoretically, we might insist upon the demonstration of definite pathologic lesions of syphilis, preferably showing the presence of the specific spirocheta, but for practical purposes we may rest content in the accuracy of our diagnosis in each suspicious case in which the serologic examinations are definitely positive, and in whom antisyphilitic therapy results not only in a general clinical improvement, but in a cessation of gastric symptoms. If this latter criterion is accepted the frequency of gastric syphilis will be considerably increased. We must remember, however, that syphilis, visceral or otherwise, and other kinds of gastric affections may occur simultaneously in the same patient, and each be independent of the other; or cases in whom the gastric condition may be functional and not organic, but secondary to syphilis, as notably seen in the gastric crises associated with syphilis of the spinal cord. Being a tertiary lesion, it is far more likely to become manifest during the middle decades of life, may affect both sexes, and, as stated above, may occur as a result of both congenital and acquired infection.

Pathologically, the disease may show any one of the following forms:

1. A diffuse gastritis, affecting the glandularis and submucosa.

2. Syphilitic ulcers, single or multiple, frequently assum-



ing serpiginous forms, and having ragged overhanging edges and a smooth base.

3. A diffuse infiltration of the gastric wall, which histologically must be distinguished from linitis plastica (unless these two conditions are one and the same, as many clinicians believe), from a diffuse scirrhus carcinoma, and from a diffuse infiltration of a tuberculous type.

4. Pyloric stenosis.

5. Gumma, which may or may not give rise to a palpable tumor.

From a histologic standpoint, the findings are practically those of tertiary syphilis occurring elsewhere. There may be diffuse round-cell infiltration, connective-tissue infiltration, frequently a general arrangement in the form of tubercles, and often, but not invariably, furnished with giant cells with the nuclei usually situated eccentrically. There may be areas of coagulation necrosis in the centers of such tubercles, but they show an indifferent tendency to the coalescence seen in tuberculosis. There may be endotheloid as well as lymphoid and connective-tissue hyperplasia. One of the most constant features is the obliterating endarteritis, which in part may account for the caseation necrosis due to lack of vascularity, and which, too, may furnish a second etiologic factor in the production of the ulcer form in gastric syphilis. In some instances the diagnosis can be made beyond dispute by the demonstration of the *Treponema pallidum* in tissue differentially stained.

A clinical classification can be readily built up, and made to correspond to the pathologic form.

The *symptoms* may vary as widely as the pathologic lesion, and in many cases there are no symptoms which of themselves can be considered pathognomonic. Quite commonly we see the symptom-complex of an organic disease of the stomach implicating both the motor and secretory mechanism. Aside from the comparatively few instances of motor obstruction due to syphilitic pyloric stenosis, the motor defect is much more commonly due to an extreme degree of atony associated with ectasia. The secretory defect is usually accompanied by the symptoms of a severe atrophic or sclerosing gastritis. Pyrosis is common, and is of the type seen in the

achylic states; sour eructations, together with the sense of an epigastric lump, weight, or pressure, sometimes associated with bloating, are the symptoms common to atony, together with the fermentations seen in ectasia.

In the ulcer form one of the early symptoms may be a profuse hematemesis, which is more likely to be recurrent than is common in simple gastric ulcer. In this form there is frequently pain, which commonly occurs late in the day, bears a less striking time relation to meals than the pain of simple ulcer, and is not so easily amenable to further food-taking, or to non-specific chemical therapy. There may be constitutional symptoms common to many diseases, such as anorexia, loss of weight, weakness and emaciation. Excessive thirst is not uncommon. In ordinary cases the intestinal functions are properly performed; when these functions are deranged, constipation is a frequent consequence.

The *gastric analyses* much more commonly show a marked subacidity or anacidity with a greatly diminished or absent enzyme activity, which is what one might expect to find associated with the pathologic defect of an atrophic gastritis. On the other hand, a few cases have been reported in which the hydrochloric acid content and peptic activity are normal, or even increased. An increase of endogenous mucus is generally the rule. Occult bleeding is frequently encountered, both in the gastric filtrate and in the feces. The blood examinations, when diagnostically helpful, usually show a chloroanemia, and a moderate leucopenia, with a relative increase of lymphocytes, and an absolute increase of eosinophiles. The serologic examinations generally yield a definitely positive Wassermann reaction, and the test is especially reliable when performed by the centrifuge method,<sup>39</sup> and when checked by the Hecht-Weinberg-Wassermann reaction as modified by Gradwohl.<sup>40</sup>

In cases exhibiting active symptoms the *x-ray* examination usually demonstrates some definite defect in the gastric outline.

The *physical findings* may give evidence of a severe constitutional infection, featured by anemia and cachexia, although these may frequently be lacking. Evidence of generalized syphilis may be disclosed in the teeth, tongue, gums or

pharynx; in the finding of a generalized adenopathy; in a manifest syphilitic eruption; in visible scars on the genitalia, or the scars from syphilitic ulcers on the extremities. Abdominal examination as restricted to the stomach itself may give no diagnostic evidence, but one frequently can demonstrate an atony, dilatation, or both in the widened area of gastric tympany, and the presence of secussion splashes. In some cases one may imagine the palpatory sensation of a thickened anterior gastric wall. There may be diffuse epigastric tenderness, but even in the definite ulcer cases painful pressure points are often lacking. In some cases there may be palpatory evidence of a gastric tumor, which in emaciated subjects may be visible. This is seen, of course, only in the gummatous forms, and in syphilitic hypertrophic stenosis. When a palpatory tumor is evident it may readily be mistaken for carcinoma, but under observation usually remains quiescent as to size or disappears under specific treatment. Further abdominal examination may disclose evidence of a syphilitic hepatitis or splenitis.

As in some other gastric conditions the *diagnosis* may have to be made by a process of exclusion. Particularly is this true of those individuals who give evidence of both a syphilitic infection and a gastric affection, each independent of the other. In those cases in whom a positive history of a congenital or acquired syphilis can be obtained, the diagnosis can be made a clinically sound one if the serologic examinations are positive, and there is a cessation of gastric symptoms, and a return to normal of the radiographic gastric contour after the exhibition of antiluetic therapy. Further than this, the writer cannot do better than quote some of the observations as published in Morgan's paper:

"1. That the failure to glean from the individual anything suspicious of a syphilitic taint, or an abortion, or failure to have children, or a negative Wassermann, does not prove that syphilis does, or does not, exist in that patient.

"2. A diseased condition of the stomach marked by a long duration with changeable symptoms, and which do not correspond to one or other of the well-recognized diseases of that organ, and which resist the accepted methods of treatment, should arouse suspicion of lues.

"3. Tumors involving the pylorus which do not cause stenosis are more often syphilitic than carcinomatous.

"4. Achylia or a low acidity, as occurred in all our cases, is usual in gastric syphilis; and where there is achylia with symptoms of ulcer, one is likely to have an ulcerating gumma or a superficial ulcer on a syphilitic infiltration base in the gastric wall.

"5. Diffuse syphilitic infiltration is usually easily detected by the palpating fingers, because it produces some enlargement of the stomach which, as happened in some of our cases, may not be readily recognized at operation. This may be true even when the infiltrating mass is to be detected by the röntgen ray.

"6. A tumor which does not change its size and shape over long periods of observation may be syphilitic, or a tumor which disappears under antisypilitic treatment may be presumed to be a gumma."

The *prognosis* is no more grave than is that of visceral syphilis elsewhere, and is usually good if the disease is properly diagnosed, and specific treatment is energetically carried out.

The *treatment* of gastric syphilis is practically the same as is indicated in any late secondary or tertiary lesion of syphilis, save those of the spinal or central nervous system.

The first essential is that the specific therapy should be thoroughly and energetically carried out, and the second essential is, that it should be kept within the physiologic tolerance of the individual patient. Since the introduction of our newer methods of treatment sufficient time has not elapsed to warrant the promise of a cure in visceral syphilis. Following the introduction of salvarsan and other forms of intravenous and intramuscular medication in many cases relapses have been frequently noted. It is yet to be proved whether long-sustained treatment with appropriate interruption may finally eventuate in a real cure. Nevertheless, we can be pretty well assured of promising our patients a symptomatic arrest of their disease.

As to the method of therapeutic procedure, this often becomes a matter of individual preference, as guided by personal experience. If no syphilitic treatment has ever been

given the patient, a more intensive and energetic form should be adopted. In the writer's opinion a good deal depends upon the strength of the serologic test. This means that every Wassermann that is returned 100 per cent. positive, or four plus,<sup>41</sup> should be *quantitatively* estimated, inasmuch as it forms such an important check on the effectiveness of our treatment. In the writer's experience in one case of gastric syphilis, with a palpable gastric tumor, presumably gumma, the Wassermann reaction was 506 per cent. positive (slightly over twenty plus), and with specific treatment was reduced to 35 per cent. positive (slightly over one plus), at which time the patient was operated upon for the relief of a complicating duodenal ulcer from which he finally succumbed.

In some cases treponemas are locked up in the heart of a pathologic syphilitic lesion, and on account of the devascularity attendant upon the endarteritis, the syphilitic antibodies may not have access to the circulating blood-stream. In such cases the Wassermann reaction may be negative, until a provocative intravenous injection of salvarsan has been given, or may be weakly positive to begin with, with a generally increasing positivity under treatment, until a definite point of pathologic cure has been reached; when the serologic reaction progressively diminishes in intensity.

In all cases of visceral syphilis the three forms of specific chemical therapy, either in periodic courses alone or in combination with one another, will be indicated: namely, potassium iodid, the various forms of mercury, and the various forms of arsenic. Where there is clinical evidence of a palpable gastric tumor, either a gumma or a hypertrophic stenosis, the action of potassium iodid, supplemented by or associated with the use of mercury, often results in a miraculous disappearance of the objective findings. These two remedies best serve to break down the connective-tissue barrier surrounding the gummatous lesions, and so liberate the spirochetæ, and give them or their antibodies access to the blood-stream, where they can be more effectively attacked by the intravenous injection of neosalvarsan, or, preferably, salvarsan. Potassium iodid should be given in the form of a saturated solution, in a dosage beginning with 10 or 15 drops (0.625 or 0.9375 mil) three times a day, preferably taken in



milk, before meals, and increasing the amount given 1 drop each dose until the physiologic tolerance of the patient has been reached, after which the dosage may be dropped to one-half or three-quarters this amount, and continued for interrupted periods of two weeks each for the first year, and gradually decreased, if warranted, during the second and third years, with short exhibitions thereafter as long as may be required. Together with this there should be given mercury, far preferably by deep intramuscular injection in the buttocks, in the form of either the soluble or insoluble salts, preferably the former. Such injections should be given daily in courses of from six to twelve, and then interrupted, to be resumed in a like period, and then to be alternated with intramuscular injections of the cacodylate of soda, beginning with 1 grain (0.06 Gm.), and increasing to 3 grains (0.2 Gm.). This is the method the writer prefers in such patients as show a low positive serologic reaction until the reaction becomes more strongly positive, when the use of intravenous injections of the arsenical group, salvarsan, neosalvarsan, arsenobenzol is to be begun, and given at intervals of a week or ten days until a course of three or four such injections have been made.

The objection to the use of potassium iodid, however useful it may be in the solution of gumma, lies in the fact of its disordering effect upon the gastric digestion. Furthermore, its use should be avoided, or most cautiously proceeded with, in tuberculous patients, especially in the quiescent, fibroid forms of phthisis, on account of the danger of lighting up this infection.

Likewise, the use of the preparations of mercury, either the protiodid or the biniodid of mercury, or the pill of mixed treatment, when administered by mouth should be deprecated, inasmuch as they not only upset the digestion, but the amount of absorption cannot be accurately controlled. For oral administration the writer prefers the use of calomel, in combination with bismuth subcarbonate or powdered chalk to counteract diarrhea. The advantage of intramuscular injections of mercury is therefore evident. If it is to be administered otherwise, the use of inunctions is the method next of choice. Calomel ointment is proving an agreeable and efficient substitute for the objectionably dirty "blue ointment."

It is needless to state that before beginning such a vigorous use of mercury, the mouth and teeth should be put in a state of oral cleanliness, and so maintained; should there be evidence of ptyalism or gingivitis, this drug should be discontinued for a short period, or its dosage materially reduced.

In cases in which the Wassermann reaction is relatively high, thus indicating that the *Treponema pallidum* or its specific products have access to the peripheral circulation, the use of intravenous injections of the various forms of arsenic should be begun at once, supplemented certainly by the use of mercury, and with less certainty potassium iodid. Salvarsan appears to be a little more effective than the neo-salvarsan, but its comparatively greater difficulty of administration makes it less commonly used. If there are no contraindications to its use, and if it is well tolerated, an injection should be given every week or ten days until four or five have been made, and then given once a month for the first year, once every second month through the second year, and twice a year thereafter as long as need be. During this time injections of mercury may be given at stated intervals, or a short course of intramuscular injections of the cacodylate of soda, and the periodic exhibition of potassium iodid in small doses, 30 to 60 drops (1.9 to 3.75 mils), daily.

The suggestions as to treatment outlined above represent the method that has proven useful in the writer's experience in the treatment of visceral syphilis. It should be thoroughly understood that there can be no syphilitic treatment given by rule of thumb, but that a definite plan should be adopted, and modified according to the requirements in the individual case. As stated above, the two important essentials are that it should be thoroughly adequate, and kept within the limits of tolerance of the patient, and finally that it should be maintained until the Wassermann reaction has been consecutively negative for a period of three years, whether this takes five years or the remainder of the patient's lifetime to accomplish. Only by this means can we be content with the assurance that the specific condition has been permanently arrested or cured.

Otherwise the treatment of gastric syphilis is purely symptomatic. The dietetic, mechanical, balneological, and

medicinal treatment is essentially the same as that outlined for the management of chronic gastritis, as discussed on page 763 *et seq.*

For the treatment of the gastric atony and dilatation the use of intragastric and extragastric electricity and vibratory massage is indicated, and these methods are discussed in detail elsewhere. (See p. 773.)

## TUBERCULOSIS OF THE STOMACH.

Tuberculosis of the stomach is a rare disease. Available statistics<sup>42</sup> give its frequency as occurring in 0.5 per cent. of all autopsies, and in 2.3 per cent. of autopsies made upon those cases dying of tuberculosis. If one is unable by gross and microscopic pathology satisfactorily to demonstrate the presence of tuberculous lesions in a given case, it is therefore quite impossible to hazard more than a mere guess that this disease has been present during life. The commonest form of gastric tuberculosis is an ulcer formation which is commonly solitary, but may be multiple. Such multiple ulcers are much more likely to occur in a generalized infection of the miliary type. The next most common form of tuberculous lesion is pyloric stenosis, in which overgrowths of tuberculous connective tissue have become localized in the walls of the stomach at the pyloric region, or in which a tuberculous pyloric ulcer has undergone cicatrization, with a narrowing of the pyloric lumen. In this connection we must bear in mind that gastric or duodenal ulcers, or benign hypertrophic stenosis, may, and more frequently do, occur as independent conditions in patients in whom extragastric tuberculosis can be demonstrated, and that unless a definite gastric pathology can be furnished such cases should not be classified under this category.

Other less common forms that have been described are the occasional solitary tubercles found in the stomach-walls, and tuberculous tumors closely simulating carcinoma, which are more apt to affect the pyloric region and cause obstruction.

In pulmonary tuberculosis, especially, and quite commonly in renal and intestinal tuberculosis, functional disturbances of the stomach become evident in most cases rather early. These

disturbances implicate both secretion and motility. In the order of their frequency this secretory disturbance is that of a subacidity with delayed digestion, next most commonly an anacidity, and but comparatively rarely a hyperchlorhydria. These clinical observations, while well known, have never been so clearly demonstrated as in the recent fractional gastric studies of H. K. Mohler and E. H. Funk,<sup>43</sup> which were carried out on 47 tuberculous patients.

While the functional suppression of gastric secretion is undoubtedly more common, many of such charted findings may be due to an organic change, non-tuberculous gastritis, which may proceed to the atrophic form. The commonest motor disturbance is that of atony, which may occur in an aggravated form, although it is generally of mild degree, and in consequence a secondary ectasia may develop. Where visceroptosis is a complicating feature the functional gastric disturbances are much more pronounced, and particularly as it affects motility.

While the etiologic factor of gastric tuberculosis (Koch's bacillus) is definitely known, its commonest portal of entry is still a matter of dispute. Theoretically, we may assume that the readiest route would occur by direct implantation of the tubercle bacillus at a receptive point of the gastric mucosa from the swallowing of tubercular sputum, or from the ingestion of food infected with tuberculous dust. This theoretic belief, however, is offset by our knowledge of the somewhat bactericidal action of the gastric juice plus the relatively faster emptying power of the stomach as compared with that of the intestinal tract, which accounts for the greater frequency of intestinal tuberculous ulceration. Again, this holds true from the fact that the stomach is furnished with much fewer lymphoid follicles than those of the intestines (Barchasch), in view of which infection by way of the lymph-channels must be comparatively infrequent, although it would seem to have occurred in such a manner in various reported cases. As we are now aware that subacidity is the commonest secretory disturbance, it is evident that the bactericidal efficiency of the gastric juice would be lessened in proportion to its amount and its concentration, and as the chief gastric motor error associated with pulmonary tuberculosis is that of atony, a delay

of gastric emptying power also is to be expected. Therefore, these two factors may counteract, to a certain extent, what has been stated above, and may permit more ready secondary infection of the stomach from swallowed sputum or food contaminated by the infecting germ.

The third portal of entry is by way of the blood, and the writer is inclined to agree with Arloing that this is the more common method, inasmuch as the diffuse miliary forms of gastric tuberculosis are more common than the chronic localized ulcers. Furthermore, Arloing has been able to produce tuberculous gastric and duodenal ulcers by injecting tubercle bacilli directly into the blood-stream.

The pathology of tuberculosis of the stomach is quite similar to that of tuberculosis elsewhere. Tuberculous gastric ulcers usually present a much more worm-eaten, overhanging edge than is seen in simple ulcer, and the floor of the ulcer may be necrotic, and, in certain cases, may be seen to be studded with miliary tubercles.

The *symptomatology* of gastric tuberculosis is by no means characteristic, and during life its presence can only be suspected. If there is a tuberculous ulcer the symptoms are much the same as those occurring in simple gastric ulcer, although commonly the pain is continuous, and may occur at an earlier time relation to food-taking. Not infrequently acute hematemesis or perforation furnishes the first proof of its presence. By far the commonest symptoms are those of insufficient secretory power with delayed motility, but it is well-nigh impossible to distinguish between those symptoms occurring as a result of organic disease and those functional disturbances secondary to extragastric tuberculosis.

As might be inferred from the brief review of symptoms, an accurate *diagnosis* during life can rarely be made. Direct diagnostic findings, such as the demonstration of tubercle bacilli in the gastric contents, is of but relative importance, inasmuch as it occurs so frequently in the swallowing of tuberculous sputum, and their presence by no means indicates that the stomach is directly infected. Similarly such diagnostic tests as the Calmette and von Pirquet reactions are not only unreliable, but when positive merely indicate the presence of a tuberculous focus, past or present, somewhere in the body.



In certain cases of gastric ulcer in patients in whom a definite improvement cannot be secured by a rigid medical *régime* for simple ulcer, or by the use of antiluetic measures, and in whom a tuberculous type of ulcer is suspected, this supposition may be strengthened if improvement follows the therapeutic exhibition of injections of tuberculin.

The *prognosis* of gastric tuberculosis, though grave enough, is no more so than that of generalized or local visceral tuberculosis elsewhere, except in the event of complications, such as an acute gastric hemorrhage or perforation.

The *treatment* is that of tuberculosis in general. Fresh air, a change of climate, if need be, rest, and a strict observance of the general principles of treatment of simple gastric ulcer, as already outlined. (See p. 684.) If there be an associated visceroptosis, the proper elevation of the foot of the bed should be carried out during the entire bed-rest treatment, and the use of a Rose adhesive plaster belt may be substituted for the Priesnitz bandage after the second week of treatment. Every effort should be made to build up weight by conserving the energy output, and by the use of high caloric diets, to the end that the resistance of the natural body defences be strengthened.

Treatment, preferably, should be carried on out-of-doors, on a sleeping porch, or on the bridge or grounds of a well-conducted hospital or sanatorium, and to be at all worth while should be carried on energetically for months.

The writer has been much impressed by the almost marvelous improvement that has taken place in certain cases of miliary intestinal tuberculosis, in which the stomach may, or may not, have been similarly infected, and in which an exploratory operation disclosed dense masses of adhesions with miliary tubercles of both visceral and parietal peritoneum, with or without free fluid in the abdominal cavity, and the intestines matted together by dense adhesions. Some such cases, though clearly inoperable, have been miraculously improved, apparently merely by opening the abdominal cavity and exposing it for a few minutes to the light and air of the operating room, draining out the free fluid, and replacing it by several quarts of normal salt solution, after which the abdomen is closed. The after-treatment has consisted of sev-

eral weeks of bed-rest, preferably in the open air, with plenty of sunshine and an abundance of nourishing food, and the use of mineral oils as an intestinal lubricant.

The writer has watched several such cases gain thirty to forty pounds in weight with a complete cessation of the gastrointestinal symptoms, notably an absence of gas-pains, and with no recurrence of the irregularly shaped, evanescent, gaseous tumors, the result of the abdominal adhesions, so noticeable before operation. While it is, of course, almost inconceivable that there should have occurred any real cure from an anatomico-pathologic standpoint, nevertheless, on re-examination of these patients, a year or two later, one would never suspect that the abdomen contained the state of affairs that was noted at the operating table months before.

As a rule, no further radical surgery should be attempted in such cases, with the exception of such localized tuberculous ulcers of the stomach as are freely accessible, and not bound down by adhesions, therefore admitting of radical resection. Even then it is a surgical question, still unsettled, whether the risk of disseminating a somewhat localized process may not be too great. The surgery that concerns cicatricial or hypertrophic stenoses of the pylorus becomes a matter of judgment whether a gastrojejunostomy should or should not be performed to secure better drainage. This will depend upon the amount of pyloric obstruction.

As to any specific medical therapy, there is none that can be generally applied with any great hope of success. The foundation of all treatment should be the appliance of the four fundamental principles of the treatment of tuberculosis in general: bodily and mental rest, fresh air and sunshine, abundant feeding, and the use of such expectant measures as may be indicated.

As regards abundant feeding, the writer is not in sympathy with the too prevalent custom of believing that this should consist of the three usual daily meals, with all the milk and eggs that the patient can succeed in eating, in addition. One must bear in mind that there are usually present the motor defects of atony, with secondary gastrectasia, and the customary plan of "forced feeding" usually serves to aggravate this condition. In addition, many of these patients show an

intolerance to a diet too rich in fat. The liquids should be restricted as in the treatment of atony and gastropnoia. The writer has found the following dietary useful in the management of this class of patients, although it has to be modified in individual cases.

*Breakfast.* A cooked cereal, such as farina, wheatena, cream of wheat or hominy, may be eaten with cream and sugar. Oatmeal may be allowed if very thoroughly cooked. An occasional lamb-chop or slice of breakfast bacon. Two soft-boiled or poached eggs. The soft parts of bread, crackers, or freshly made toast, may be eaten with butter. Milk, malted milk, or cocoa may be taken. It is better to avoid both tea and coffee.

10 to 11 A.M. The choice of cream and rice-water formula, or malted milk. Koumiss, kefir, buttermilk, or equal parts of milk and cream, junket, or cup-custard. One or two raw eggs may be substituted, or added to any of the foregoing. Crackers and butter.

*Luncheon or Dinner.* Chicken or fish in any form but fried, broiled squab, or the breast of guinea-hen. Broiled or boiled beef and lamb, to be run through a grinder when cooked. Milk toast. Oysters in any form but fried. Potatoes in any form but fried, preferably baked or mashed. Peas, lima beans, spinach, squash (to be put through a colander and puréed with cream), boiled rice, tender string-beans, buttered beets, creamed carrots, or the tender ends of asparagus or cauliflower, spaghetti or macaroni. A salad with plain lettuce and French dressing (with the amount of vinegar reduced) may be permissible every second day, if desired. Bread and butter. Choice of junket, cup-custard, blanc mange, tapioca, rice, cornstarch, or bread-puddings, floating island, and vanilla ice-cream, if held in the mouth until warmed to body temperature.

4 to 5 P.M. The same choice as 10 A.M.

*Supper or Dinner.* Thick soups, such as rice, sago, barley, farina, potato, or asparagus; creamed purées of beans, peas or lentils, which are to be run through a colander. No soups made from meat or meat-stocks are allowed. One or two soft-boiled or poached eggs. Bread and butter. Milk or cocoa, and the choice of any of the above desserts, except ice-cream.

Before retiring the choice of the foods allowed at 4 P.M.

*Avoid.* Fried, greasy foods; pies, cakes, candy, hot cakes, mustard, pepper, vinegar, pickles, onions, coarse breads, and all fruits.

The writer recommends the following prescription, which was a favorite of the late John H. Musser, as a symptomatic remedy for the relief of the symptoms of epigastric weight, pressure, and fullness, with sour or gaseous eructations, symptoms common to gastric atony and ectasia with fermentation:

R	Creosoti .....	gr. ss to j (0.03 to 0.06 Gm.).
	Spts. chloroformi .....	℥iiss (0.14 mls).
	Spts. ammoniæ aromatici ....	℥v (0.3 mls).
	Sodii bicarbonatis .....	gr. v (0.3 Gm.).
	Liq. sodæ et menthæ (N.F.)	
	q. s. ....	℥j (30 mls).
M.	S.: One teaspoonful (3.75 mls), to be taken in a little water one-half to one hour after meals.	

This combination has proven very effective in relieving the increased intragastric pressure, relaxing the cardia, and permitting readier belching, with symptomatic relief. Any of the stomachic or blood-building tonics may be adopted, according to the symptomatic indication, and their use is often most helpful.

For the direct treatment of the associated atony and dilatation, with or without gastritis, in addition to the use of hygiene and dietetics, lavage supplemented by intragastric or extragastric faradism, or sinusoidalization, will prove extremely beneficial, unless contraindicated for reasons of general debility.

A few words in regard to the biologic-therapeutic agent, tuberculin. Since first introduced by Robert Koch in 1890, tuberculin has enjoyed a many-colored reputation, at times brilliant, and for many years somber. At the end of a ten-year period following its introduction, its use was generally discredited. This was due, no doubt, to the massive dosage then commonly employed, which produced severe reactions, and numerous reports were published stating that it was a dangerous weapon, and had a tendency to hasten tubercu-

lous (pulmonary) lesions to caseation and cavity formation. About 1905, largely due to the efforts of Sir Almroth Wright, its use was begun again, but on a minimal dosage plan. Since then the results secured have been undoubtedly improved. It has had far greater vogue in Europe, especially in England, than it ever enjoyed in this country. Indeed, some of our most eminent experts on tuberculosis have never used it, nor do they express faith in its efficiency. The writer has had no personal experience with it as a therapeutic agent, and should therefore be guarded in indorsing its use. When given it should be administered by the subcutaneous method (certainly not by mouth), and in minimal ascending doses, by one who has thoroughly studied such authorities as Riviere and Morland,<sup>44</sup> and who, possessed of a thorough knowledge of the indications and contraindications for its use, has become familiar with the fundamental principles of its technic.

### PYLOROSPASM.

As its name implies, this condition is one in which the musculature of the pylorus, already more powerful than that occurring at any other point in the stomach, suddenly undergoes local spasm, which occludes the lumen of the pyloric exit and results in its obstruction. This obstruction is often only temporary, but may result permanently if gastric atony or ectasia secondarily develop.

The opening and closing of the pylorus is controlled by two factors: (a) A central nervous mechanism. The motor accelerating fibers of the pylorus are largely derived from the vagi and the inhibitory fibers from the cervical splanchnics, and these or their combination generally make up the primary mechanism of respectively closing and opening the pyloric orifices. (b) A local chemical or mechanical reflex exerted at the pylorus. As a chemical factor it is found that increased concentration of the gastric juice frequently leads up to or accompanies pylorospasm, causing motor delay; and, conversely, relaxation of the pylorus occurs with increased motility in the achylic states, except when due to cancer. Again, as a mechanical factor the ingestion of coarse or badly comminuted foods results in pylorospasm due in part to the



irritating effect such coarse foods have upon a localized pyloric erosion or fissure. Also it is known that greasy or oily foods, while their exit from the stomach is normally delayed, nevertheless result in pyloric relaxation, and permit duodenal regurgitation. This fact was made use of in the method of recovering duodenal contents after the ingestion of olive oil or cream; and therapeutically olive oil is useful in controlling pylorospasm.

Clinically, pylorospasm is divided into those cases occurring as a primary neurosis due to irritation of the vagi (vagotomy); secondly, pylorospasm due to a localized irritation, as in pyloric or duodenal ulcer, erosion or fissure; and, thirdly, pylorospasm, *reflexly* induced by irritation at other points of the midgut or its derivatives, notably irritation of the biliary apparatus of the appendix.

Pylorospasm has also been noted in cases of gastroptosis, prostatic disease, and lesions of parts of the female productive apparatus. The last-named, together with a primary neurosis, constitute by far the rarer types of the condition. It is most commonly associated with irritative lesions, ulcer, erosion and fissure, located at or near the pylorus, and next most commonly caused reflexly by inflammatory irritation of the biliary apparatus or the appendix.

The *symptoms* of pylorospasm are quite typical. Usually at the height of digestion, one to two hours after a mixed meal, the patient experiences a sudden spasmodic cramp-like pain in the epigastrium at the pyloric point, which may radiate around either costal margin or to the back, is soon followed by nausea, and in cases with hyperacidity a burning epigastric distress, with or without acid regurgitation. If the condition is due to a gross dietetic error, in which the pylorospasm is a protective mechanism of nature to prevent the duodenal or intestinal reception of an improper chyme, vomiting may occur or be induced after which the spasmodic pain of pylorospasm disappears, and the other sensations of epigastric distress gradually wear away. With chronic pylorospasm, where pyloric obstruction has continued for long, the symptoms common to atony and ectasia will make their appearance—post-prandial epigastric sense of weight and pressure, with bland, sour, or acid regurgitations and eructations.

During the acute attack one will usually find a tender point in the epigastrium, approximately 1 inch (2.5 cm.) to the right of the median line, and from 1 to 2 inches (2.5 to 5 cm.) above the navel. In patients with very flaccid abdominal walls one may feel the spasmodically contracted pylorus, and if gentle palpation is continued the pylorus relax is frequently palpable, and the little tumor will disappear from beneath the palpating finger. Indeed, if the patient is greatly emaciated and the abdominal wall is thin, this spasmodic contracture and relaxation of the pylorus may be visible through the abdominal wall, and has frequently been observed at the operating table during a laparotomy. The writer saw an interesting case in Paul Cohnheim's clinic, at Berlin, in which the spastic pyloric tumor could be produced at will, and readily palpated, after forcible pressure had been made over the sternomastoid muscles to irritate hypertonic vagi.

Where pylorospasm is reflexly produced from gall-bladder disease it is important to determine the physical findings common to such condition, such as stiffening of the right costal arch and tenderness to palpation, or forcible percussion over the gall-bladder, with rigidity of the upper right rectus muscles. In cases of chronic dyspepsia with pylorospasm due to an appendiceal irritation, pressure over McBurney's point frequently gives a pain definitely referred directly to the region of the pylorus, with occasional palpatory evidence of pylorospasm. After the acute symptoms have subsided epigastric soreness and tenderness to pressure persist for several days.

The chemistry of the stomach may show no abnormality, particularly when interpreted in the light of a single extraction at sixty minutes, but by the fractional method it is more common to find a normal first hour cycle with hyperacidity becoming evident during the second hour, and frequently hypersecretion demonstrable during the third, fourth, and fifth hours.

The *prognosis* for prompt symptomatic relief is very good, except in cases due to primary neurosis which may prove very intractable and are curable only by the restoration of a proper nervous balance. In cases reflexly dependent upon primary surgical condition, palliative medicinal measures may give

symptomatic relief, but final cure cannot be expected until the aseptic scalpel has done its work.

### TREATMENT.

In pylorospasm due to a primary neurosis the underlying treatment must rest upon building up the general nervous system to a greater plane of stability. To this end *hygiene* is of prime importance. An abundance of fresh air by day and night, moderate exercise in the open-air, adopting some form of exercise that is attractive to the individual, are to be advised. Hydropathic measures are useful, such as the morning cold bath, followed by a salt friction rub. In this connection a simple method to adopt is to soak a rough turkish towel in a pail full of brine for a few minutes, allow it to dry rapidly so that the salt is crystallized out and becomes enmeshed in the towel, and then vigorously rub the surface of the body. Alternating hot and cold spinal douches, with the stream directed particularly over the third to eighth dorsal vertebrae to stimulate the inhibitory action of the splanchnic nerve supply to the stomach, are helpful hydropathic supplements. Intragastric electricity, particularly the sinusoidal, and to a less extent the galvanic, current, has proven useful in certain cases. The external electrode should be connected to the negative pole, and be in the form of a pad sufficient to cover the third to the eighth thoracic vertebrae. Likewise, a maneuver suggested by Abrams (*loc. cit.*) of sustained pressure by means of a bi-forked pressor instrument, placed over the spinal column between the third and fourth thoracic vertebrae, will relieve pylorospasm promptly, but is a much more effective procedure in acute dilatation of the stomach. (*Cf.* p. 824.)

Nerve sedatives, such as the bromids, sumbul, valerian, and the like, may be given for short periods and then should be followed by one of the stimulating tonics.

In pylorospasm, when due to a local irritation, such as ulcer, erosion, or fissure, the treatment must be more direct. In acute pylorospasm due to gross dietetic error, and in all cases in which the pylorospasm results in acute pyloric obstruction, prompt lavage with alkaline solutions to rid the stomach of its offending irritating contents is extremely bene-

ficial, after which, if the pylorospasm still continues in its acute form, nothing gives more prompt relief than a hypodermic of  $\frac{1}{4}$  grain (0.015 Gm.) of morphin with  $\frac{1}{100}$  of a grain (0.0006 Gm.) of atropin sulphate, after which further injections of atropin sulphate in a dosage of  $\frac{1}{200}$  to  $\frac{1}{300}$  of a grain (0.0003 to 0.0002 Gm.) should be given every second or third hour until the symptoms are under control or the point of physiologic effect has been reached. Local applications of heat applied to the epigastrium are very grateful to the patient, but in pylorospasm due to ulcer with a history of recent bleeding ice-cold abdominal applications should be used instead. (*Cf.* p. 697.) For one or two days no food should be given by mouth, and nourishment should be given by enemata and the use of proctoclysis.

In chronic pylorospasm due to a chronic calloused pyloric ulcer, especially where there is motor obstruction and secondary hypersecretion, surgical interference is indicated and a pylorotomy or a pyloroplasty are more effective procedures than a gastrojejunostomy. Where the pyloric obstruction is due to a pylorospasm with secondary inflammatory edema and not to a chronic stenotic process, such as a cicatrix, medical measures may be carried out and given a thorough trial before surgery is attempted. To this end the general plan of the ulcer cure should be adopted.

When pylorospasm is reflexly produced from irritative lesions of the gall bladder, if the surgical indications for operative interference are not definitely clear, palliative medicinal measures may be adopted, in addition to the steps outlined above. Urotropin (hexamethylenamin) may be given in a dosage of 5 grains (0.3 Gm.) three or four times a day, and a teaspoonful (3.75 mls) of Carlsbad salts may be given in a tumblerful of hot water on the fasting morning stomach; also, one may give 5 grains (0.3 Gm.) of the inspissated bile salts thrice daily, two hours after meals.

When pylorospasm is due to appendiceal disease operative interference affords the only means of permanent relief, and any palliative treatment is merely postponing the inevitable. In all operations in the upper abdominal zone for a condition in which pylorospasm is a symptom, whether a local irritative lesion is or is not found at the pylorus or duodenum, no opera-

tion is complete unless both the gall-bladder region and the appendix are inspected and corrective surgical measures adopted. In certain cases the writer has seen pylorospasm continue after a gastrojejunostomy for pyloric or duodenal ulcer, and only cease when a pathologic appendix was removed at a subsequent operation. When pylorospasm is reflexly produced by gastropptosis, with a dragging pressure exerted on a firmly fixed pylorus or duodenum, the symptom usually disappears when the stomach is elevated to a better position by a proper abdominal support.

### PYLORIC OBSTRUCTION.

The etiologic factors of pyloric obstruction are herewith given in their relative order of frequency:

1. Cancer or sarcoma of the pyloric exit, causing obstruction from within; or primary cancer or sarcoma of the neighborhood viscera, causing pyloric obstruction from without.

2. Benign agencies. Pyloric obstruction secondary to cicatricial contraction, from a pyloric ulcer, or following the ingestion of corrosive substances.

3. Pylorospasm, due to a local irritation of the stomach (ulcer, fissure, etc.), with inflammatory edema, resulting in obstruction.

4. A pericholecystitis or pericholangitis with adhesions to the pylorus or duodenum, or, conversely, peritoneal adhesions to the neighborhood viscera, from a perigastritis or a localized upper abdominal peritonitis.

5. Congenital pyloric stenosis or a stenosing gastritis.

6. Benign tumors of the stomach obstructing the pylorus, chiefly polypi or adenomata.

In the early stages of this condition the most conspicuous *symptoms* are those of motor insufficiency common to atony or ectasia, giving a sense of epigastric pressure and heaviness occurring at varying times after eating, but usually reaching their height one to two hours after taking food, depending largely upon the amount of food ingested. This epigastric weight and discomfort are accompanied by belching or food regurgitation with temporary relief. Nausea may be complained of, but as a rule it does not occur until accumulative



food retention has taken place. The frequency of vomiting will of course depend upon the degree of motor insufficiency. In the lesser grades so much food passes through the pylorus that the retained residue is comparatively small, and may require several days for a sufficient amount of food to collect that provokes voluntary or induced vomiting. As the obstruction increases, less food is able to pass the pylorus at any given meal, and retention or collective vomiting may occur daily or every second day. When this point is reached fermentative processes usually have begun, and, in addition, the patient will show progressive loss of weight, due to inanition, through failure of food to reach the intestines and be absorbed. Thirst is usually complained of, and the degree of retention can frequently be gaged by the diminution of the daily urinary output. When the urine output has decreased to 500 mils (16 f̄5), in the absence of a primary nephritis, the obstruction has reached a very severe grade. In hyperacid cases, pyrosis and acid regurgitations are usually complained of, and if there is an associated hypersecretion the amount of fluid in the retention vomiting will be materially increased.

The chemical and microscopic examinations in pyloric obstruction due to cancer show the hydrochloric acid elements generally diminished or absent, and the presence of lactic acid, the Oppler-Boas bacillus, and occult bleeding can usually be demonstrated. In pyloric obstruction due to benign causes the hydrochloric acidities are likely to be higher, and butyric and acetic acid, with high fermentation tests, and sarcinæ are usually present. The diagnosis and the degree of pyloric obstruction can best be determined by a series of motor test-meals.

The *prognosis* is always grave, but will vary according to the etiological factors involved.

#### TREATMENT.

With the exception of pylorospasm with obstructive inflammatory edema, and with the possible exception of a localized upper abdominal peritonitis, particularly if chronic or subsiding, the treatment is entirely surgical. Such surgery may consist of a pylorotomy, a partial gastrectomy either with or without a gastrojejunostomy, or a gastrojejunostomy

alone, the operative choice being dependent upon the individual operative mechanics, the comparative risks in each type of operation and the age and condition of the patient. This particularly applies to pyloric obstruction due to malignancy and to congenital pyloric stenosis.

When due to ulcer or to inflammatory adhesions associated with biliary diseases, the choice of the most favorable operation must be modified to meet the requirements of the individual case. Benign tumors of the stomach that have a tendency to develop from the mucous membrane and obstruct the gastric lumen, the adenomas and polyps particularly, rarely need radical resection, and many of the cases reported have been sufficiently localized to be removed through a gastroscope. In the writer's opinion, however, this type of operative treatment is more heroic, and carries a greater operative risk, even when practised in the most experienced hands, than does direct surgery following laparotomy.

Where pyloric obstruction is due to pylorospasm with inflammatory edema, and if not due to reflex surgical lesions of the gall-bladder or appendix, the medicinal management, as discussed on page 799, should be given a thorough trial for at least a week before surgical interference should be considered.

The degree of pyloric obstruction may give the indication for the urgency of operative interference. This naturally does not apply to cancer, the radical cure of which primarily depends upon immediate operation. Where cancer can be reasonably excluded, a few days of delay, while palliative measures are being tried, will not materially increase the later operative risk to the patient, and the writer is coming more and more to the belief of the need of giving patients who require gastric surgery, particularly those showing motor insufficiency, a week or ten days of preliminary medical treatment with bed-rest, daily lavage, and the use of external abdominal applications. By a few days of such treatment, in association with the other measures detailed elsewhere (*Cf.* p. 799), one can usually decide in a given case of pyloric obstruction how much is due to pylorospasm with inflammatory edema, and whether under the circumstances a continuance of such medical management would be warranted.

### CONGENITAL PYLORIC STENOSIS.

In view of the still continued dispute as to its etiology, congenital pyloric stenosis might better be termed "hypertrophic pyloric stenosis of infants."

The nature of this dispute revolves around two theories, each of which has been supported by loyal adherents. The first of these theories is that the hypertrophy affecting the circular muscular fibers of the pylorus is congenital. It is caused by abnormal proliferation of this muscle layer during fetal life, and in the few days or weeks that elapse after birth, prior to the development of symptoms, and secondary to this congenital hypertrophy a pylorospasm develops simultaneously with the appearance of symptoms. The supporters of this theory of congenital development base their belief largely upon the ground that a degree of hypertrophy, such as exists in most cases, could not have occurred as a work hypertrophy secondary to pylorospasm, in the few days or weeks that have elapsed since birth. The opponents of this theory, however, point to the absence of any recorded cases of overdevelopment of the pyloric musculature seen in autopsies on the fetus.

The second theory concerns itself with the premise that the condition is primarily a pylorospasm, reflexly or locally produced (*e.g.*, pyloric ulcer, erosion, or fissure) with a secondary hypertrophy of the pyloric muscles as nature's attempt to overcome the obstruction. The opponents of this theory believe that if such were the case there should be evidence of hypertrophy of the longitudinal as well as the circular muscle coats, and they ask why, in analogous cases of spasm of the sphincter ani, due to anal ulcers or erosions, there does not occur secondary hypertrophy of the rectal muscles on the proximal side. It will probably clarify the situation to assume that these two theories are conflicting simply because they are not attempting the description of the same clinical entity.

There has been an attempt to classify the cases into two groups:

1. Cases of primary pylorospasm with secondary hypertrophy. These show milder gradation of symptoms, and have

largely been considered medical, inasmuch as many respond satisfactorily to medical measures designed to allay primary pylorospasm and pylorospasm secondary to pyloric ulcer.

2. Cases of congenital hypertrophy of the circular muscular fibers of the pylorus, with added pylorospasm occurring as a secondary factor. Such cases rapidly develop a much more serious aspect, and are generally to be considered a surgical problem for the reason that they do not prove amenable to successful medical management.

From a strictly etiologic standpoint such a classification is not justifiable, if one presents the subject with a title of *congenital* pyloric stenosis. Nevertheless, such a distinct clinical entity undoubtedly exists in which the pyloric hypertrophy is prenatal in origin. One of the best arguments in favor of this is that cases have been observed (Holt) in which the pyloric hypertrophy was found *post-mortem* from two to five years after gastro-enterostomy, in children who subsequently died from other causes. It is scarcely conceivable that a work hypertrophy, secondary to spasm, would persist so long after the primary cause has been removed. It should also be remembered that even if the hypertrophy is strictly congenital, it may occur in all degrees from the mildest to the most severe.

To Beardsley, who in 1788 reported a case with *post-mortem* findings, certainly belongs the credit for the first accurate description of this disease. Half a century later additional cases were reported, but the attention of the medical profession was not drawn to the frequency of this condition until the publications of Hirschsprung, of Copenhagen, in 1888, and it is to him that we principally owe our modern conception of this disease.

The most authoritative *résumé* of the entire subject has been recently published by Emmett Holt,<sup>45</sup> to whom the writer cordially acknowledges his indebtedness for the surgical data in connection with this section.

At operation or at autopsy the pylorus is found to be hypertrophied, and appears as a cartilaginous cylinder extending from the pyloric ring 1 to 2 inches (2.5 to 5 cm.) toward the gastric side of the pylorus, and pouting into the duodenum, much as the cervix uteri projects into the vagina. On section the muscular wall is seen to be greatly thickened and the

hypertrophy limited to the *circular* muscular fibers together with the hypertrophy of the pyloric mucosal folds. There is often secondary edema. The color is a pearly white, and is notable for its bloodless appearance; the pylorus and pyloric ring are stenotic, and may barely admit the passage of the smallest probe, largely because of the secondary hypertrophy of the mucosal folds with the superadded edema. The rest of the stomach is usually found to be considerably dilated, as the consequence of a secondary process.

The great majority of these cases run true to form and show a marked similarity of *symptoms*. The history obtained is exceedingly important, and is usually that of a breast-fed infant, most commonly a male, born in perfect health and remaining so for several days or several weeks, rarely exceeding ten. According to Holt's observations, the onset of symptoms is likely to occur between the second and third weeks of life. After taking the breast well such an infant suddenly begins to vomit, and this occurs daily. The vomitus at first is usually small in amount, and consists entirely of the ingested food with no evidence of biliary regurgitation. In a few days, according to the rapidity with which secondary dilatation develops, accumulative or retention vomiting occurs, and can be readily determined from the fact that the quantity of the ejected material is considerably greater than the amount given at the last feeding. Vomiting usually occurs shortly after the feeding, and is probably due to secondary pylorospasm, which is almost invariably an associated feature in these cases. The stomach is highly irritable, and peristalsis is active until atonic dilatation has occurred. The vomiting is projectile in type, may be ejected through the nostrils as well as the mouth, and may project for a distance of several feet. Shortly after vomiting has begun the infant begins to lose weight, at first slowly, and later very rapidly, frequently reaching a state of extreme emaciation in a very few days, if the condition is unrecognized or unarrested. The bowels, as a rule, become very constipated. This is of diagnostic value, inasmuch as in most other conditions of infantile vomiting there is the associated diarrhea of enteritis. The microscopic examination of the fecal residue shows a diminu-



tion of food elements, and the urine becomes scanty and high-colored owing to the lack of retention of ingested fluids.

The objective findings are likewise characteristic. Test-meals of 2 or 3 ounces (30 or 60 mls) of breast milk or condensed milk may be recovered one or two hours later in practically the full amount, indicating pyloric obstruction. For this purpose Holt recommends the use of a soft rubber catheter, attached to one end of a T-tube inserted through the cork of an ordinary laboratory specimen bottle, to the other end of which is attached a suction tube, by means of which gentle aspiration of gastric contents can be made by direct mouth suction. The most important objective finding is the observation of waves of gastric peristalsis, running downward from left to right. This is an almost invariable finding, although it may require considerable patience and frequent observation before it is noted in some cases, and while pathognomonic is by no means indispensable to the making of the diagnosis. Its presence depends to a certain extent upon the degree of emaciation. Such a tumor is felt as a cartilaginous ring or cylinder about the size of a hazel nut, at a point usually just above and to the right of the navel.

The *diagnosis* in most cases should be easy, inasmuch as the symptoms are so uniformly characteristic. In the order of their diagnostic importance, Holt classifies the symptoms and objective findings in the following order:

1. The history if intelligently given and taken. A history of a healthy born, usually breast-fed male infant, with the abrupt onset of vomiting of a projectile and progressive type; and the appearance of symptoms within a few days to a few weeks after birth, but much more commonly between the second and third weeks of life.

2. A determination of abnormal gastric retention indicative of pyloric obstruction.

3. Visible peristaltic waves, provided they are typical.

4. The presence of a palpable tumor.

5. The associated symptoms of wasting, constipation, and scanty urine.

Diagnosis by means of *x-ray* plate or fluoroscopic examination is not essential, inasmuch as little more in the way of

direct confirmation can be gained which cannot be deduced from the history and clinical observation, and because these measures add a certain element of risk, and cause unnecessary fatigue.

The differential diagnosis is concerned itself chiefly with these two conditions:

1. Pylorospasm secondary to duodenal or pyloric ulcer, in which there may be seen the visible peristalsis. These cases, however, give quite a different history. There is not the evidence of persistent pyloric obstruction, a greater tendency to hypersecretion exists, visible or occult bleeding in gastric filtrate or in the feces is usually demonstrable, and unless the pylorospasm is continuously persistent considerably less wasting from inanition occurs.

2. Gastric indigestion or gastro-enteric indigestion of various types with protracted vomiting and subsequent emaciation. Here the diagnosis is made by the exclusion of the other characteristic symptoms, common to congenital pyloric stenosis. The vomiting is not so persistent or extreme and there is usually no evidence of a high-grade pyloric obstruction, the characteristic constipation is usually replaced by a bloody and mucous diarrhea secondary to enteritis, and the stools are sour, fermentative, and greenish-yellow instead of the hard and dry, as in pyloric stenosis due to absence of fluids in the intestines.

In view of the advances in diagnosis and treatment the *prognosis* has become much less grave than was formerly justifiable. The mortality has decreased about 25 per cent. with improved surgical technic and a perfected follow-up treatment.

### TREATMENT.

The character of treatment in the final analysis resolves itself entirely into a matter of judgment whether any given case should be managed medically or surgically. In recent years a few series of cases have been published in which many successful recoveries have been reported by the adoption of many perfected medical plans. Nevertheless, the mortality here ranges from 30 to 50 per cent. Furthermore, it is probably true that most of these reported medical recoveries occurred

in cases exhibiting milder grades of the disease, or, indeed, cases of primary pylorospasm or secondary to pyloric or duodenal ulcers, fissures, or erosions. If this be a fact the latter group cannot be classified as congenital pyloric stenosis. In the writer's opinion, even such cases as give classically characteristic symptoms cannot be as accurately diagnosed as those cases which come to the operating or autopsy table.

If a medical plan is adopted, it should be continued only to such a point as shows a persistent and steady improvement. Cases that do not show an immediate gain in weight, or that progress badly should not be temporized with until progressive weakness and emaciation mitigate against later successful operative interference. On the other hand the writer agrees with Koplik,<sup>46</sup> that unless a surgeon of experience and technical skill is available it may be wiser to continue the medical management. This view must, however, be somewhat modified from the fact that at the time of Koplik's publication a gastrojejunostomy was considered to be the operation of choice, and required approximately from one-half to three-quarters of an hour for its performance; whereas the operation of choice today is that devised by Rammstedt, which has proven vastly more efficient, can be carried out with great celerity, and has reduced the surgical mortality by nearly 30 per cent. This operation will be discussed in later paragraphs.

The essentials of medical treatment may be summarized as follows:

1. *Feeding.* If the infant is breast-fed it is by far better not to wean it, but it is of an advantage to pump the breast and feed the baby at hourly intervals in small amounts by means of a medicine dropper. It is needless to say that the quality of the mother's milk should be promptly analyzed to determine its suitability. If the baby has been bottle-fed on modern scientific plans, and still shows persistent loss of weight, it is better to substitute a properly selected wet-nurse. It should be seen that both the breast milk and any modified feeding formulas should be relatively low in fat.

2. *Lavage.* The stomach should be lavaged twice a day before feedings by means of a soft catheter and glass funnel.

3. *Bowels.* The bowels should be moved exclusively by enemas, and this should be followed by proctoclysis with a

solution of decinormal soda bicarbonate, to which may be added  $2\frac{1}{2}$  to 5 per cent. of glucose.

4. *Local Applications.* Heat should be applied constantly to the abdomen in the form of hot, moist compresses, over which may be placed a soft felt pad electrically heated.

5. *Weight.* The infant should be weighed daily, and the weight carefully recorded. If any good is to be accomplished by a medical management, it will be promptly seen in the cessation of the vomiting and a prompt gain in weight.

### CARDIOSPASM.

Cardiospasm, once thought to be a rare disease, is being recognized much oftener on account of our increasing familiarity with its symptoms and the technical measures necessary for its direct diagnosis.

The etiology of cardiospasm is varied, many causes having been advanced, all doubtless capable of proof in certain cases. The following causes have been suggested:

1. Primary cardiospasm. (Meltzer.)
2. Primary esophagitis. (Martin.)
3. Primary atony of the esophageal musculature. (Rosenheim.)
4. Functional disturbance of the innervation of the esophagus due to paralysis of the vagus causing simultaneous spasm and atony of the musculature of the esophagus. (Kraus.)
5. Congenital disposition. (Fleiner, Zenker, Luschka and Sievers.)
6. Kinking at the hiatus esophagei. (Plummer.)

Bassler has recently published an article in which he puts forward the view that cases exhibiting obstruction at the lower end of the gullet, of the type which we have hitherto thought to be cardiospasm, are not cardiospasm, but rather "a spasm of the esophageal opening of the diaphragm due to contraction of the muscular fibers of the crura, which contracts the esophageal opening by drawing the central tendon of the diaphragm against the front of the esophagus or contracts it at the sides."

In support of this view, which is based on a dissection of

5 fresh cadavers and Röntgen-ray observations of 7 cases of cardiospasm, he seems able to prove (1) that "the lower extremity of the esophagus or cardiac orifice of the stomach have no or only a faintly developed sphincter," and (2) that the stricture is almost always epicardial, usually occurring at a distance of one vertebra above the cardiac orifice of the stomach, which corresponds to the esophageal opening of the diaphragm.

Bassler offers no etiologic factors tending to produce this diaphragmatic contraction of the esophageal opening. While his paper throws a new light upon the subject, it would appear to be one concerning proper nomenclature, affecting somewhat the pathology, but not altering appreciably the symptomatology or therapy of what we now call cardiospasm.

Under normal conditions, we know that food on entering the esophagus requires from seven to ten seconds to pass into the stomach; about one-seventh to one-quarter of this time is required for food to pass down to the cardiac portion of the esophagus, where it remains for several seconds until the cardiac sphincter relaxes, and the food passes into the stomach by the peristaltic contraction of the circular and longitudinal fibers of the esophageal muscles. We have ample proof of this (1) in the Röntgen-ray studies of Kronecker and Meltzer, Cannon and Moser, and others (2) by the common personal observation of experiencing the sudden aching pain of momentary duration felt back of the sternum in its lower third on swallowing too hot or too cold foods, such as soup, coffee, or ice cream, and (3) by the *post-mortem* evidence, after the ingestion of corrosive poisons, that the greatest amount of erosion in the esophagus occurs at its lower third in the neighborhood of the cardia, where contact with the corrosives has been longest sustained.

While there is this normal delay of foods at the cardia, this varies also with the character of the food, its temperature and chemical concentration, the extremes of all these causing a variable inhibition of the dilating mechanism of the cardia.

Idiopathic or primary cardiospasm occurs frequently, as is seen so often in hysteria and in those of neurotic temperament and tendencies, and is here purely functional; but if of frequent occurrence it permits more and more retention of foods,



often irritating in character and composition, for too long a time in the neighborhood of the cardia, predisposing to the development of esophagitis of varied types, erosions, fissures and ulcerations. Thus a vicious circle is produced, the local inflammation disposing to cardiospasm, and the spasm permitting of unduly long retention of foods at the cardia, thereby increasing the esophagitis.

The commonest forms of cardiospasm are *primary cardiospasm* of a purely functional type, and constituting a local neurosis, or a manifestation of a general neurosis, and *primary esophagitis*; but when both cardiospasm and esophagitis can be demonstrated simultaneously, it is extremely difficult to decide which is the primary lesion.

Of the other etiologic factors, congenital disposition may be an active element in certain isolated cases. Indisputable proof of this, however, should be furnished by the history and the absence of all other etiologic factors before one could feel reasonably safe in assigning this as the cause. Likewise, primary atony of the esophagus, while it may occur rarely, is probably also a less common factor in the etiology. Plummer,<sup>47</sup> to whom we are largely indebted for our better understanding of this condition, concludes from a study of his cases, that primary atony of the esophageal musculature is of rare occurrence, stating that in his cases "the almost invariable history of spasm at the onset, followed in the later period by the evidence of dilatation—that is, retention of food in the esophagus—is most convincing evidence that the spasm precedes the dilatation, and that primary atony is rare." Functional disturbances of the innervation of the esophagus, due to the paralysis of the vagus, and kinking at the hiatus esophagei, also appear to be unusual etiologic factors.

There are undoubtedly many cases of cardiospasm so slight as not to give rise to any *symptoms*, and in such instances the diagnosis can be established only by mechanical means. The first subjective symptom usually volunteered by the patient is a sensation of discomfort felt behind and usually to the left of the sternum. This is variously described as a dull, aching pain, that throbs, burns, cuts, or has a sense of pressure or weight, as if something had lodged low down in the gullet. These symptoms occur only during the ingestion of food, and

at first may be of short duration, with periodic remissions during which the patient is able to eat freely and without dysphagia.

During this period the esophageal musculature is sufficiently strong to overcome the spasm and to permit of the entrance of food into the stomach with only momentary delay. As the condition progresses, compensatory hypertrophy of the musculature must develop to overcome the increasing obstruction, and here a second symptom makes its appearance, namely, the regurgitation of foods from the esophagus into the mouth, very shortly after their ingestion, due to the overactive contracting efforts of the esophageal musculature. The regurgitated foods in this stage may be both liquid and solid, returning in practically the same condition as when eaten, and not unpleasant in taste or odor. The majority of ingested food is passed through the cardia slowly, but nevertheless surely, so long as the hypertrophied muscle is competent to overcome the obstruction. Gradually the muscles tire under their extra load, and rupture of the muscle bundles takes place with a resultant dilatation. At this stage the regurgitation of food may be temporarily less frequent, and occur at somewhat longer intervals after it is swallowed. As the dilatation becomes more extreme, the esophageal capacity becomes greater, and capable of retaining larger quantities of food, which are likely to be regurgitated only when the patient is lying down, stooping over, or during a paroxysm of coughing. The dilatation of the esophagus in time may become extreme, with a capacity well over a pint (500 mls). Since the propulsive power of the esophageal muscle is lacking, food can only pass into the stomach slowly even when the spasm has been relaxed, and in proportion to the weight of the column of food in the esophagus, assisted by gravity. The liquid portion of the meal usually passes more rapidly, seeping through the solid portion so as to leave a dense pultaceous, often foul-smelling mass, usually incorporated with tenacious mucus, which gives rise to a continual sensation of pressure and fullness behind the sternum, with occasional difficulty in breathing, due to pressure on the trachea. As food products are retained for longer and longer periods within the esophagus, fermentation and decomposition, chemical and bacterial, take

place, and this results in secondary esophagitis. At this stage the condition of some patients is truly deplorable; they suffer continually with a sense of burning pressure back of the sternum; they are able to eat only small quantities of food at a time, and their total amount of food ingested and assimilated is so small that they lose weight rapidly; and if not relieved may develop a profound cachexia and die, literally of starvation.

The symptom-complex is usually so characteristic as to suggest the *diagnosis*, although I must confess that it is not always so easy as it sounds.

The direct diagnosis can always be made by the use of an esophageal bougie, preferably of the Plummer type, very often by means of the stomach tube, or by fluoroscopic study and Röntgen-ray plate analysis.

In the early stages, with the use of the esophageal bougie or of the stomach tube, it will be found that an obstruction to the further passage of the instrument in adults is met with at about 16 or 17 inches (40.6 or 43.2 cm.) from the incisor teeth. The instrument can be passed readily until this point is reached, when an elastic-like obstruction is met with, which, under firm and steady pressure, usually gives way, and permits of the passage of the bougie or stomach-tube.

During the periodic remissions the obstruction will not be met with, or at times the bougie or tube seems about to pass when it is suddenly gripped in the spasm of the contracting muscle, excited by the instrumentation. Great care should be practised in attempting to pass the instrument beyond the obstruction until the diagnosis of cardiospasm is definitely made to the exclusion of diverticuli, a kinking at the hiatus, malignant esophageal stenosis, or external pressure by an aneurysmal sac or mediastinal tumor.

To rule out these differential possibilities the Röntgen-ray had best be employed first, to disclose the presence of a thoracic aneurysm, mediastinal growth, the presence of diverticuli, and the irregular outlines of a carcinoma infiltrating the lower end of the esophagus and causing a stenosis. After aneurysm and mediastinal tumor have been eliminated, it is safe to proceed with further instrumentation. If either a diverticulum, a kinking of the hiatus esophagei, or a carcinomatous stenosis is

suggested by the Röntgen-ray examination, it is best to make use of the esophageal bougie devised by Plummer, which consists of a series of olive tips, which are attached to a stout whale-bone staff. The olives are perforated from about their middle to the tip. Six yards (548.64 cm.) of thread are then swallowed by the patient, preferably half of this length one afternoon and the other half the following morning, which permits of the thread passing well down into the upper coils of the intestines and becoming fixed so firmly that strong traction can be made on the proximal end emerging from the mouth. The olive tip is then passed over this thread, and by means of traction on the thread the olive tip can be safely guided through the cardia. By varying the amount of traction the sound can be introduced into a diverticulum, and its depth and size determined.

The diagnosis of cardiospasm likewise may be confirmed by a test which I believe has hitherto not been reported, namely, by esophageal lavage. With the tip of the stomach-tube in the esophagus, at a point just above the obstruction, water is allowed to run in from a graduated glass tank. It will be seen to run much more slowly than if the tube were in the stomach. From 100 to 500 mls, (27½ to 1 pt.), according to the amount of esophageal dilatation, will run in slowly, but evenly, until the flow suddenly stops, and the level of the fluid in the graduated glass tank begins to oscillate slightly. At this point the water is allowed to escape through the out-flow tube, and without changing the position of the stomach-tube it will be seen that the amount recovered is equal to the amount introduced. When the capacity of the esophagus has been reached, if instead of opening the outflow tube the fluid is allowed to remain in the esophagus, by its weight, assisted by gravity, it will cause the cardiospasm to relax. This will occur in a varying number of seconds, according to the degree of the spasm, and will permit some of the water in the esophagus to pass into the stomach, thereby allowing an additional flow from the glass tank, which will then usually proceed in a uniform way until recovered by introducing the tube *farther into the stomach*. While for purposes of demonstration, this last step may be permissible, it is never wise to distend the esophagus to its point of capacity, as it exaggerates the al-



ready existing atony. The essential point in diagnosis by this method is the ability to recover *from the esophagus itself* an amount of fluid equal, or nearly so, to that introduced. This is not possible in diverticulitis or in pressure stenosis of the cardia.

*It is also of prime importance to determine the presence of a secondary or complicating esophagitis.* This is possible by means of the esophagoscope, but its use is so formidable to the patient that the writer prefers to make the diagnosis by the examination of esophageal sediments obtained by a method published elsewhere. It is necessary to determine the extent and kind of this complicating esophagitis because it indicates the proper application of the principles of treatment, *for as long as the inflammatory condition exists so long will the cardiospasm persist*, notwithstanding efforts directed toward the latter to the neglect of the former.

Properly treated patients do well, except possibly those instances of cardiospasm secondary to an esophagitis caused by the action of corrosive poison, which heal with difficulty, and result in contractions due to scar tissue, which, though healed, may still predispose to spasm.

#### TREATMENT.

In the earlier cases of cardiospasm of the primary type, relief usually may be obtained by the administration of anti-spasmodics, such as belladonna and atropin, pushed to the limit of tolerance and with due regard to a possible neurotic etiologic factor. The regulation of proper hygiene, and the use of hydrotherapy and exercise, preferably in the open air, should be advocated. If these measures do not suffice, esophageal bougies may be used, or the spastic cardiac ring may be dilated by means of dilators of the types suggested by Plummer and Bassler. When there is a concomitant esophagitis, measures should be adopted toward allaying this before proceeding to the treatment of the cardiospasm. Suitable measures are the lavaging of the esophagus with medicated solutions, best determined and controlled by the character of the esophageal sediment. If the esophageal erosion or ulceration has become secondarily invaded by bacteria, one can use germicidal solutions, such as potassium permanganate, silver



nitrate, argyrol, etc., until the bacteria have disappeared from the inflammatory desquamation, when blander solutions, such as boric acid or normal salt solution, are to be substituted. The use of an autogenous vaccine prepared from cultures grown from the esophageal sediments will facilitate recovery from the severer types of esophagitis. If the inflammation is sharply localized in the form of ulcerations or erosions, healing medicaments may be directly applied by means of long applicators introduced through a small bore esophagoscope or through a rubber tube just long enough to reach the incisor teeth to the lower end of the gullet. When the condition has progressed to the stage of esophageal dilatation and atony, the use of intra-esophageal electricity is indicated, preferably with the sinusoidal current or the faradic current, by means of a suitable intragastric electrode. The negative pole should be attached to the electrode within the esophagus, and the positive pole to the external electrode in the form of a hand sponge, which is to be carried over the transverse processes from the seventh cervical to the third thoracic vertebra, and over the sternomastoid muscles, particularly the left, to stimulate the vagus. Before turning on the current the patient should drink a small glassful of water to serve as a better conductor of electricity and to prevent burning by direct contact. The duration of each treatment should not exceed ten minutes, and should be given daily in severe cases until improvement is noted. In those cases showing progressive loss of weight, due to inanition, it is important to arrange the diet in the form of liquids, the total caloric value of which for twenty-four hours should be over 3000. This can be accomplished by the liberal use of milk and cream, olive oil, butter, egg-nog, soft-boiled or raw eggs, and non-stimulating broths.

In the very late cases that come under observation during the extreme stage of starvation asthenia, it is perhaps better to do a preliminary gastrostomy, and to feed directly through the stomach, thus trying to build up the strength of the patient before proceeding to the other treatments. Apropos of operative procedure, Bassler (*loc. cit.*) suggests, "that in intractable cases the approach to the site and cause of the stricture had best be made by the safer abdominal route rather than through the thorax, and that an operation which has to do

with the division of the crura, either at their insertions or the bisection of two of the inner portions of both at the back of the gullet or some point in the esophageal opening is worthy of consideration."

It is necessary to observe certain patients over a long period of time. Relapses from primary cardiospasm, properly treated, are comparatively uncommon, but relapses due to exacerbations in any residual esophagitis are more frequent.

### GASTRECTASIS.

Gastrectasis is an enlargement of the stomach, coincident to, or associated with a diminution of its motor expulsive power which may be either relative or absolute. The motor defect must be present if one is to differentiate these cases from megalogastria, in which the size of the stomach is abnormally enlarged, but in which the motor function remains efficient. In gastrectasis the motor insufficiency may be absolute in the sense that the muscular power of the gastric wall has depreciated to the extent that gastric contents cannot be propelled into the duodenum within normal time limits, or it may be relative, as in those cases in which there is pyloric obstruction, and in which the gastric musculature, although hypertonic, is nevertheless unable to overcome the obstruction.

Gastrectasis may occur in both an acute and chronic form, the latter being probably quite as common as the former is rare.

Gastrectasis is most easily differentiated from pyloric obstruction, especially in the chronic form, by the fact that while there is marked motor delay during the digestive periods, the stomach is able to empty itself completely overnight. This is the rule, although occasionally in the more severe forms the fasting morning stomach shows overnight retention.

**The Acute Form of Gastrectasis** (acute dilatation of the stomach). Brinton is accredited with the first description of this condition, more than sixty years ago, but it remained for Fagge, twenty years later, accurately to describe its clinical features. Since then over 300 cases have been reported in the literature; but its frequency is far greater than this, if one considers the large number of unpublished cases. While it is a

distinctly unusual condition from a comparative standpoint, doubtless many cases, especially those of mild degree, have escaped recognition.

Form 1. Dilatation of the stomach.

Form 2. Dilatation of the stomach and first portion of the duodenum.

Form 3. Dilatation of the stomach, the entire duodenum and occasionally the jejunum.

The known etiologic factors are somewhat varied, but most of them can be combined under the heading of an acute toxemia, which may locally cause a paresis of the gastric musculature, or may paralyze the motor fibers of the vagi and the cervical sympathetics supplying the stomach. This toxemia may arise during the course of acute infections, prominent among which may be mentioned typhoid fever, pneumonia, miliary tuberculosis, and occasionally cardiorenal disease. More common than these are sudden toxemias of still obscure causation occurring as post-operative manifestations, especially following operations upon the stomach and mid-gut, although quite frequently it has been noted in operations involving the lower abdominal zones. A contributing factor to such toxemias are the post-narcotic effects of prolonged ether or chloroform anesthetics.

Mechanically, gastrectasis can be produced by an acute traction upon the duodenum, by angulation of the duodenum by the mesenteric vessels (mesenteric ileus); by deformities of the spine acting similarly and perhaps also interfering with the motor fibers of the peripheral portion of the vagus; or by displaced abdominal neighborhood viscera. Again certain cases have been noted after traumatic injuries to the head in which the motor nerves have been supposedly injured. Acute dilatation confined to the stomach alone can be caused by simultaneous spasm of the pylorus and cardia with a sudden increase in intragastric tension. Those cases in which the dilatation involves the segment of the gut below the duodenum, are necessarily very severe and may predispose readily to a fatal issue.

Again, dietetic errors (*e.g.*, gastric dilatation following the ingestion of foods that have rapidly undergone fermentation of high degree) have been occasionally reported as etiologic

factors. If so, they are far less common than those mentioned above.

In the course of acute infectious diseases, usually at their toxemic height, or within a few days after operation or prolonged anesthesia, the *symptoms* may be ushered in with acute epigastric pain which may be somewhat localized to the right of, or at, the median line in the neighborhood of the pylorus or duodenum. This is rapidly followed by profuse and continuous vomiting. Symptoms of shock may rapidly appear or may be delayed for a few hours. Unless observed carefully the *initial period of shock may be overlooked*. Indeed, the symptoms may be so closely allied to those of an acute intestinal obstruction, or to an acute perforated viscus, that close observation and rapid differential diagnosis must be applied to prevent primary operative interference. In some cases the condition is not ushered in with acute pain, but instead a gradually developing epigastric or left hypochondriac soreness and a dull sense of pain may be associated with the other symptoms.

Vomiting is most conspicuous. If associated with dietetic errors the first vomitus consists of the fermenting gastric chyme, but otherwise it is likely to be somewhat grumous brownish material flecked with bile-stained mucus. Later on biliary vomiting of large amounts of light or dark greenish fluid indicates duodenal dilatation and pyloric relaxation. Where the jejunum is likewise involved a different kind of *brownish green* vomitus is recovered which has the characteristic odor of, and may be microscopically diagnosed as, fecal vomiting. The one most characteristic feature of this vomiting is its excessive amount and its fluidity, which is partly due to a transudation from the gastric and duodenal mucosa. Later on, as retching continues occult bleeding can be demonstrated in the vomitus, and later blood-stained flecks of mucus appear.

In the severer cases well-marked gastric tetany may be observed, and this was a conspicuous symptom of one of the writer's cases of acute dilatation of the stomach, associated with the acute gastric crises of syphilis.

The *objective findings* are characteristic, and inspection of the abdomen usually discloses a visible and palpable tumor

conforming to the position of the stomach, whether normally situated or abnormally displaced. In one patient it occupied a vertical position as an enormous bologna-shaped tumor, with the identical surface topography of the descending colon. According to whether the contents of the stomach are gaseous or fluid, there will be a widening of the area of gastric tympany, or a widened area of percussion dullness replacing gastric tympany. The knees are usually held drawn up against the abdominal wall in an effort to reflex the lower abdominal zones. Diffuse epigastric or left hypochondriac palpatory soreness or tenderness can be made out. If there is peritoneal irritation, muscle rigidities may be noted. After the condition has been present for a varying length of time, usually a few hours, the pinched features of the patient rapidly assume the Hippocratic facies, and the objective evidence of circulatory shock may be marked.

In the severe cases no difficulty is encountered in making the *diagnosis*. It is in the lesser grades that a keen observation and interpretation of the symptoms must be developed. In any case in which vomiting of the type described occurs in the course of an acute infection, or is prolonged beyond the first few hours after operation or anæsthesia, the suspicion of this condition may be strongly entertained. It is signally important that the differential diagnosis of this condition should be separated from acute intestinal obstruction, volvulus, intussusception, and acute arterio-mesenteric ileus, which may secondarily produce acute dilatation of the stomach. Indeed, it is only the results of the first few hours of energetic treatment that in some cases serves to make this differential diagnosis.

The prognosis of gastrectasis, whether moderate or severe, is serious, and in the latter is essentially grave, and will depend largely upon the early recognition of the condition, and the energetic skill with which it is treated.

#### TREATMENT.

In all cases whether primarily produced by tractional pressure on the duodenum, as in mesenteric ileus, or secondarily caused by the weight of the fluid contents sagging down a parietic gastric wall, thereby causing pyloric or duodenal kinking, the first essential therapeutic requirement is the adoption



of the proper postural attitude, to assist in the prompt mechanical emptying of the stomach. To this end the foot of the bed should be elevated from 12 to 20 inches (30.4 to 50.8 cm.), and the patient turned from the dorsal position, or, if his strength permits, placed in the knee-chest position. Assisted by attendants, after this position has been maintained for a short time, he should be placed in the right or left antero-lateral abdominal position. Which of these two positions best mechanically fulfills its purpose possibly cannot be told until both have been tried in the individual case. As a general rule, the left antero-lateral abdominal position allows the weight of the fluid-filled stomach to fall away from the duodenum and to straighten out any kink at this point. Again, this position has been fluoroscopically determined as best suited to the rapid emptying of the stomach.

To be placed in this position the patient should be turned on his ventral surface, with the head to the left and slightly overhanging the side of the bed. The weight should be placed on the left antero-lateral aspect, the right arm being curled under the head or stretched out across the bed, while the left arm may be allowed to hang down, supported by a chair, or it may be placed parallel to the left side of the body. To aid in throwing the weight on the left side, the right half of the body is supported by a series of low pillows, from neck to heels. In some cases it is of assistance to place a small bolster-type pillow under the abdomen at about the level of the navel. Such a pillow should measure from 12 to 16 inches (30.4 to 40.6 cm.) long and 6 to 8 inches (15.2 to 20.3 cm.) in diameter, and is readily constructed from a pound roll of absorbent cotton wrapped in a bath towel and fastened with adhesive tape.

The next essential point of treatment is prompt lavage, which should be thorough, frequently repeated, or, indeed, made continuous. The writer recommends the following procedure:

After thoroughly washing out the stomach with a large sized stomach-tube (32 to 34 F.), it is withdrawn and replaced by the smaller calibrated duodenal tube, introduced either through the mouth or through the nares, and, after reaching its proper level in the stomach, strapped securely

to the patient's cheek by adhesive plaster. To avoid traction upon the nostrils or pharynx, the tube should be firmly fastened by passing it through a safety-pin pinned to the pillow or mattress at a point 6 to 8 inches (15.2 to 20.3 cm.) from the patient's face. The proximal end of the duodenal tube is attached to one of the horizontal arms of a small calibrated glass T-tube, to the other horizontal end of which is attached rubber tubing running to an outflow pail, and the vertical end of the T-tube is attached by means of rubber tubing to an irrigating tank of 1 or 2 liters (quarts) capacity suspended 2 to 3 feet (60.8 to 91.2 cm.) above the patient's head. A pressure clamp should be placed over the rubber tubing between the irrigating tank and the T-tube, and a second one between the T-tube and the outflow pail. By alternately releasing pressure upon the tube from the irrigating tank and that running to the outflow pail, the patient's stomach can be continuously and gently lavaged and aspirated by siphonage.

The lavaging fluid should be warmed to the body heat, and should consist of plain water,  $\frac{1}{10}$  normal solution of soda bicarbonate, or normal salt solution. The writer has likewise found a 1 to 3 solution of alkalol useful on account of its bland alkalinity, and it may either be introduced through the irrigating tank or directly into the duodenal tube by means of a 2- or 3-ounce Triumph syringe. The writer cannot emphasize too strongly the value of this continuous method of lavage. The small tube is readily tolerated by the patient, and overcomes the objection of the frequent introduction and removal of the large-sized stomach-tube.

Where hypersecretion is evident in such cases, especially if associated with pylorospasm, the hypodermatic use of atropin sulphate in repeated doses of  $\frac{1}{200}$  to  $\frac{1}{300}$  of a grain (0.0003 to 0.0002 Gm.) will be of material advantage. If there is marked gastric hyperesthesia, 1 or 2 ounces (30 to 60 mls) of olive oil containing 2 to 3 per cent. of anesthesin may be introduced through the duodenal tube, or 5 to 10 grains (0.3 to 0.6 Gm.) of chloretone, dissolved in 2 or 3 ounces (60 or 90 mls) of water, may be similarly introduced. The patient may be allowed to suck cracked ice, although none should be swallowed. If there is pronounced nausea, 2 to 3 drams (7.5 to

11.2 mils) of brandy or *crémé de menthe* may be added to the cracked ice. The writer has lost confidence in the efficiency of cerium oxalate in controlling nausea or vomiting. It is essential that all foods should be withheld by mouth until all nausea has ceased and the stomach has become definitely retentive, and when feeding is resumed the dietary should conform to the general plan of the ulcer cure. In the meantime nourishment is to be supplied by rectal enemata and by the use of proctoclysis with a  $\frac{1}{10}$  normal soda bicarbonate solution, to which may be added 5 per cent. of glucose, to assist in overcoming a starvation acidosis.

The mouth may be swabbed out with a mixture of 1 ounce (30 mils) of glycerin mixed with the juice of one lemon or one orange, or with orange albumin. Supportive measures should be carried out to combat collapse by the use of hot water bottles or electric pad warmers, until secondary pyrexia has occurred, when ice or cool sponges will be grateful to the patient. Strict attention to nursing details are quite as essential to recovery as is the direct medical care.

Where the above measures do not result in favorable improvement, the writer wishes to particularly emphasize a method that has served well in 5 cases within his personal knowledge. It is a mechanical maneuver<sup>48</sup> and consists of making of pressure by means of a bi-forked metal pressor instrument (devised by Abrams) over segmental areas of the spinal sympathetic nervous system. For the relief of acute dilatation of the stomach, a firm degree of pressure should be exerted for from thirty to sixty seconds with the pressor instrument placed across the spinal column in the interspaces between the third and fifth thoracic vertebræ. Within a certain number of seconds explosive eructations of gas occur, or a sudden rush of gaseous or fluid contents can be heard, passing ostensibly through the pylorus or duodenum and into the jejunum, with prompt disappearance of the gastric tumor. In some cases this maneuver must be repeated every fifteen or twenty minutes for one or two hours before the condition is permanently under control. Abram's method of spondylotherapy should by no means be urged to supplant the other methods of treatment outlined above, and, indeed, possibly it should not be resorted to, in the light of our present knowledge, until all other measures have been exhausted.

As to just what interpretation can be made of the startling results of this procedure, a final answer cannot at present be given. The writer is not prepared to accept Abram's<sup>49</sup> explanation of this so-called "stomach contraction reflex," but would like to suggest the following hypothesis as a plausible explanation of the effectiveness of this treatment: The simultaneous cardio- and pylorospasm may be the result of a lawless or over-stimulated vagus (a vagotony), which the inhibitory action of the thoracic sympathetic fibers to the stomach is powerless to overcome until mechanically stimulated by forced pressure. It is probable that the true explanation will be lifted from the veil of obscurity when our knowledge of the states of vago- and sympathetico-tony and atony is more complete. Nevertheless, the writer is prepared to say that this method has proved successful in some cases where all other methods had failed. Abrams also claims that a contraction reflex of the stomach can be obtained by strong percussion over the transverse spines of the first, second, and third lumbar vertebræ.

To the writer this maneuver certainly appears worthy of a careful trial, although it should not supplant other therapeutic measures. It does not seem capable of doing lasting harm, and may give such unexpectedly brilliant results as will warrant its use.

Among other medicinal agents that may be employed, may be mentioned the use of eserine sulphate in a dosage of  $\frac{1}{50}$  to  $\frac{1}{100}$  of a grain (0.0013 to 0.0006 Gm.), in combination with  $\frac{1}{30}$  of a grain (0.00216 Gm.) of strychnine, which may be repeated every three or four hours until intestinal peristalsis is excited.

For the relief of tympanites, when results cannot be obtained by simple enemas followed by colonic irrigation, an alum enema may be substituted, using 1 ounce (30 Gms.) of powdered alum to a pint (500 mls) of water. If this does not serve to empty the bowels and relieve tympanites, elaterin may be given by hypodermic injections in a dosage of  $\frac{1}{4}$  to  $\frac{1}{2}$  grain (0.016 to 0.032 Gm.), reinforced by  $\frac{1}{30}$  of a grain (0.00216 Gm.) of strychnine to overcome the depressing action of the elaterin. Both elaterin and eserine should be used cautiously, and never if the patient is in a state of exhaustion.

Other medicinal agents are not indicated, except in the way of supportive expectant management.

If these measures fail, but little hope can be entertained from surgical interference. In former years gastrojejunostomy was frequently practised, but the results were not favorable. In cases due to a kinking of the duodenum from adhesions, or in those with evidence of intestinal obstruction lower down, exploratory laparotomy may be justifiable, but to be of any use it must be undertaken promptly.

### GASTRIC CRISES OF CEREBROSPINAL SYPHILIS.

Spinal or cerebrospinal syphilis is comparatively common, and its incidence has been considerably widened since the demonstration of the *Treponema pallidum* in the meninges and in the nerve tissues of the spinal cord and brain.

In most cases the infection of the nervous system takes place during the late primary and early secondary periods, and thus may potentially affect the nervous system in all cases. That all cases are not so infected warrants the belief that there are various strains of the treponema, each possessing selective affinity for certain tissues. A racial immunity is likewise suggested if we consider the rarity of tabes dorsalis in the Chinese and the negro, both of which races are conspicuously saturated with syphilis.

In tabes dorsalis it is more than of passing interest that the history frequently shows an inconspicuous or atypical primary lesion, and the absence or mildness of secondary symptoms and lesions. This might mean either an infection with a strain of treponema having an affinity only for the nervous system, and an inability to colonize in other viscera or body tissues, or it might mean that the very mildness of the primary and secondary lesions may have caused the individual so infected little recognition of the seriousness of his disease, so that its treatment is relatively or absolutely neglected, until the onset of cerebrospinal symptoms.

Among the most interesting and important of these early symptoms of cerebrospinal syphilis are the visceral crises, which generally make their appearance in the preataxic stage,



These visceral crises may be laryngeal, bronchial, cardiac, gastric, intestinal, renal, rectal, and genital.

It is the purpose here, however, to discuss briefly the symptoms, diagnosis, and treatment of the gastric crises.

The *symptoms* of gastric crises consist of sudden seizures of upper abdominal pain, preceded, accompanied, or followed by vomiting, and associated with various disturbances of secretion. These attacks usually occur with extraordinary suddenness, may strike down the individual while in apparent good health, and frequently with no premonitory symptoms. The crisis may last for several days, occasionally for two weeks or more, and usually ceases as spontaneously as it occurred, with a sudden restoration to the patient's normal state of health, despite the severity of the attack.

Historically, several authorities, Graves, Romberg, Grube, and Delamarre reported cases exhibiting this symptom-complex which antedated Charcot's publications of 1868 and the years following, but by universal consent to Charcot is given the credit for his masterly presentation of the symptoms and pathology of the gastric crises occurring in *tabes dorsalis*, for which clinical picture he coined the term "*crises gastrique*."

For a few years there ensued diagnostic confusion, resulting in published reports of visceral colics with nausea, vomiting, hemorrhages, diarrhea, etc., but associated with various purpuric skin lesions or edemas, which would place them among the group of visceral crises occurring in angio-neurotic edemas and erythemas.

Sainton and Trenck have described in great detail six different varieties of gastric crises. Suffice it to say that there may be attacks exhibiting a great variety of symptoms, varying both as to duration, frequency and severity. There may be mild attacks featured only by vomiting, with absence of pain and absence of secretory disturbances. There may be attacks of great severity, with agonizing pain and persistent vomiting, first of gastric contents, later duodenal contents, and still later jejunal contents.

The greatest characteristic of any attack, *no matter of what variety, is the startling suddenness with which it is ushered in, and its equally abrupt termination.* Even after a severe and

protracted crisis of a week or longer, when it is over the patient becomes immediately hungry, wishes to eat, and unless his case be complicated by organic gastric disease, for instance, such as ulcer, the stomach ceases to be irritable, and does not occasion any digestive disturbances.

During a severe attack, where pain is conspicuous, it may be agonizing, and cause the patient to assume all sorts of bizarre positions to gain relief. While an attack is in progress the abdomen is usually retracted, except in those cases complicated by gastric or duodenal dilatation; is often extremely tender to palpation, especially in the epigastrium; the muscles are often held rigid, and if difficult vomiting has been persistent, the thoracic and abdominal muscles maintain a soreness which may last for several days after the attack has subsided. In the severe attacks the amount of prostration may be very great, partly due to continual retching in the vomiting types associated with acute gastric dilatation, and partly due to cardiovascular failure, which may imminently threaten a fatal collapse. Probably some cases of this latter type are due to direct spirochetal colonization in the heart muscle.

In another case the premonitory symptom was of another type. For several days before the attack there was noted a striking increase in the amount of bile-stained fluid vomited each morning from the fasting stomach. If this fluid could be gotten rid of by vomiting, the symptoms might be aborted. If emesis could not be secured, even by induced retching, the sensation of double-retrosternal and epigastric lump (to be described) would begin, and within a few hours an attack would be under way. This observation led me to empty this patient's fasting stomach each morning by duodenal tube, until he had learned to do so himself. This measure has not only brought him the greatest amount of relief, but has permitted eight months to elapse since his last attack.

As stated earlier, pain, vomiting, and various disturbances of gastric secretion make up the usual triad of this condition. Yet, there is another group of symptoms to be regarded as of great importance. Not uncommonly there is a complaint of lower thoracic and upper abdominal sense of fullness and pressure seen early in some attacks, which may progress to an unbearable sense of a ball-like lump felt behind the lower

third of the sternum, a sense of a "fixed lump" that seems impossible either to get up or down, and a sense of a second lump, referred to the right, mid, or left epigastrium, along a line just above or at the level of the navel.

This sensation of lumps may suddenly disappear, and the epigastric pressure-fullness may be relieved, often only temporarily, by explosive belching of gas, or by the passing of gas from stomach to intestines, or by expulsion of gas from the rectum. This type of crisis may be similar to that described by Fournier of the flatulent variety of gastric colic, in which, without apparent cause, there occur attacks of loud eructations of odorless and tasteless gases for several days, but unaccompanied by vomiting.

Again, there is a mild form of gastric crisis, featured by sudden easy and apparently causeless vomiting, independent entirely of food-taking, or the character of the food. This vomiting is spontaneous, painless, may occur several times a day for several days, and the vomitus may be gastric or biliary, may be practically odorless and tasteless, or may be sour, fermentative, bitter or rancid, depending upon the state of the gastric motility and chemistry, and yet, apparently, is not primarily dependent upon these as causative factors.

Gastric crises appear in tabes with considerable frequency; probably one out of every third or fourth case will present this complication, and, in addition to this, the gastric crises frequently appear as the *initial* symptom of tabes. With the history of sudden onset and symptoms of acute abdominal pain associated with vomiting, many a tabetic suffering with gastric crises has been rushed to the operating table for an unwarranted laparotomy. The literature on the subject is replete with instances of this sort. Nuzum, in his study of 1000 tabetics, found that 97 (nearly 1 in every 10) had been operated upon under the mistaken diagnosis that the gastric crises were an expression of some form of abdominal surgical disease, and yet nothing was found intra-abdominally to account for the symptoms. This can be avoided by a thorough examination of the nervous system of such patients.

The *diagnosis* in a good many cases is easy, and in others exceedingly difficult. If the patient is observed in his first attack of gastric crisis, and especially if it presents the initial

symptom of a cerebrospinal syphilis, a positive diagnosis cannot be made without a serologic, chemical, and cystologic examination of the spinal fluid. Of course, one who has seen a sufficient number of such cases to have had the peculiar symptomatology impressed upon his attention may correctly hazard a guess.

A carefully taken history will naturally throw much light upon the relative importance of the diagnostic possibilities concerned in any given case. If a preceding luetic infection is admitted, gastric crises *should always be considered*.

While the usual trinity of symptoms consists of pain, vomiting, and disturbances of gastric secretion, they are by no means always present in any given case; one or two may be lacking, and even if all three are present such symptoms may occur in many other conditions.

The examination of the spinal fluid is the *sine qua non*. In the majority of cases of spinal syphilis it will show a pleocytosis, an excess of globulin, and a positive Wassermann reaction. Until this has been made, it is often wiser to maintain an attitude of masterful inactivity, or watchful waiting, if we would avoid the humiliation of an unjustifiable exploratory laparotomy. Fordyce and others endorse the value of the Lange or colloidal gold test of the spinal fluid to distinguish true paresis from simulating types of cerebrospinal syphilis.

Where gastric crises do not appear as the initial symptom of spinal syphilis, the diagnosis is often clarified by an examination of the nervous system. Irregularities in the size and outline of the pupils, or, still more significant, an Argyll Robertson pupil; a positive Romberg sign; the absence of one or both knee-jerks (Westphal's sign), or a break in the arc of either of the deep reflexes (absence of the Achilles tendon reflex, etc.); a thoracic zone of hyperesthesia or anesthesia will make the examination of the spinal fluid yield largely corroborative testimony. Transitory ocular squint or lesions of the auditory nerve are frequently monosymptomatic forerunners of tabes, and should receive more than passing attention.

The *prognosis* is generally serious, and often extremely bad.

## TREATMENT.

The writer's method of treatment during *acute attacks* is as follows:

Absolute bed-rest; elevation of the foot of the bed, 8 to 12 inches (20.3 to 30.4 cm.) to guard against dilatation of the stomach, which is not uncommon. Absolutely no food by mouth is allowed. Paraffin wax should be chewed every 2 hours. The patient may suck cracked ice, to which may be added two or three teaspoonfuls (7.5 or 11.2 mls) of brandy or *crème de menthe*, if there is pronounced nausea. The mouth may be swabbed out with a mixture of 1 ounce (30 mls) of glycerin and the juice of one lemon. If vomiting is a feature of the attack, the stomach should be emptied by means of a duodenal tube and syringe aspiration. An analysis of the aspirated fluid to some extent will determine the chemistry of the lavaging fluid. If such an analysis cannot be done at once, plain water is the safest lavaging fluid. After the stomach has been gently washed with  $\frac{1}{2}$  to 1 liter (1 pint to 1 quart) of fluid by alternate syringe injection and aspiration, the tube is left *in situ* at the proper level, and securely strapped to the patient's chin or cheek by adhesive plaster. The proximal end of the tube is attached to one of the horizontal arms of a small T-tube, to the other horizontal limb of which is attached rubber tubing running to an outflow pail, and the vertical end of the T-tube is attached by means of rubber tubing to an irrigating tank of 1 to 2 liters (1 to 2 quarts) capacity, suspended 2 or 3 feet (60.8 or 91.2 cm) above the patient's head. A pressure clamp should be placed over the rubber tubing between the irrigating tank and the T-tube, and a second one between the T-tube and the outflow pail. By alternately releasing pressure upon the tube from the irrigating tank and the tube running to the outflow pail, the patient's stomach can be continuously and gently lavaged and aspirated by siphonage. The lavaging fluids should be warmed to body heat, and should consist of plain water, 1:10 normal solution of soda bicarbonate, or normal salt solution to which 5 per cent. of glucose may later be added, should there be evidence of acidosis. The value of this continuous method of lavage cannot be emphasized too strongly. As a rule, it is well



tolerated, usually controls the vomiting, and prevents cardiac strain or injury to the gastric mucosa from continuous painful retching. If there is evident hypersecretion, hypodermics of atropin sulphate may be injected subcutaneously in dosage of 0.0006 gram (gr.  $\frac{1}{100}$ ) every hour until three doses have been given, and then every three hours; or after one or two injections have been given from 30 to 60 minims (1.9 to 3.7 mls) of the tincture of belladonna may be added to each liter (quart) of the irrigating fluid. This will control both the hypersecretion and relieve or prevent pylorospasm and its consequent pain. If there is marked gastric hyperesthesia, 1 or 2 ounces (30 to 60 mls) of olive oil containing 2 to 3 per cent. of anesthesin may be introduced through the duodenal tube, or 0.3 to 0.6 gram (5 to 10 gr.) of chloretone, dissolved in 60 to 90 c.c. (2 to 3 ounces) of water, may be similarly introduced.

For the relief of the double-point spasm, and especially if gastric dilatation has taken place, the writer wishes particularly to call attention to a method which he has found useful. It is a mechanical maneuver, and consists of the making of pressure by means of a bi-forked pressor instrument (devised by Abrams) over segmented areas of the spinal sympathetic nervous system. For the relief of acute dilatation of the stomach, if caused by simultaneous double-point spasm, with a resultant rise of intrasegmental tension, a firm degree of pressure should be exerted for from 30 to 60 seconds, with the pressure instrument placed across the spinal column in the interspaces between the third and sixth thoracic vertebræ. Within a comparatively few seconds the patient will begin to belch explosively, as the cardia relaxes first, or a sudden rush of fluid or gaseous contents can be heard passing ostensibly through the relaxed pylorus or duodenum into the jejunum, and simultaneously the sense of pressure, fullness, and the painful sensation of a double fixed lump is relieved. Should there be gastric dilatation, the area of gastric tympany rapidly becomes smaller. This maneuver may have to be repeated several times before a successful result is obtained.

If gastric dilatation is pronounced, in addition to the elevation of the foot of the bed and the other measures suggested,

the patient should be placed in the right lateral abdominal position, and a small bolster pillow should be put just below the lower border of the stomach, to raise it to a higher level, and thus to prevent an aggravation of the condition by duodenal or mesenteric kinks or angulations.

These measures control the nausea, vomiting, hypersecretion, gut-spasm, gastric dilatation, and to some extent the pain. For really severe pain, such as that due to irritation of the posterior dorsal nerve roots, nothing short of morphin has proved thoroughly satisfactory. If the pain is moderate, some of the drugs suggested above may control it.

The circulation should be carefully watched during the acute attack, and if the systolic blood-pressure falls below the pulse-rate the patient is in need of stimulation.

Feeding by mouth should not be resumed until all nausea has ceased, and the stomach becomes definitely retentive, and when begun the food should be given in liquid form, frequently, in small amounts, and gradually increasing, as in the ulcer cure (*q.v.s.*).

In the meantime nourishment is to be supplied by rectal enemata and by the use of proctoclysis with 1:10 normal soda bicarbonate solution, to which may be added 5 per cent. glucose, and if need be 2.0 to 4.0 grams (30 to 60 gr.) of sodium bromid to allay restlessness and nervous apprehension.

When convalescence is established, and the patient's hunger has returned, it is wise to remember Rosenheim's caution not to overload the stomach, on account of the danger of producing motor errors.

External applications to the epigastrium of wet compresses, either hot or cold, give variable pain relief in the same individual; sometimes one will help, sometimes the other. Other forms of counterirritation, such as the mustard plaster or fly-blister, may be tried.

Cerium oxalate has been extensively tried, both for the relief of pain and for vomiting, with varying endorsements. Lockwood states that it has been worthless in his hands. Ostankow reported good results from its use, which led Basch to try it in 18 cases. He gave it in doses of 0.1 gram ( $1\frac{1}{2}$  gr.) every two to four hours during the attack, and three times a day between the attacks, and found that it did not

in any sense relieve the pain, but helped in controlling the nausea and vomiting. He likewise tried the effect of antipyrin, as recommended by Gowers and Zippert, giving it in doses 0.25 gram (4 gr.), repeated hourly for four doses, and if no symptoms of circulatory depression occurred he increased the dose 0.5 gram or 0.1 gram (7 to 15 gr.). He came to the conclusion that it acts as a general sedative, especially in those cases with pain, but had no effect in controlling vomiting.

Carrying out a suggestion by Oppenheim, Basch tried the subcutaneous injection of 0.002 gram ( $\frac{1}{30}$  gr.) of nitrate of strychnin in five cases, which showed no improvement after either cerium oxalate or antipyrin, and in two cases secured relief from pain and restful sleep, but no satisfactory effect in the other three.

Hunt mentions the use of injections of cocain into the epidural and subarachnoid spaces of the spinal canal, as suggested by Oppenheim, and a similar use of alcohol and stovain, as recommended by Levy and Pope. He also mentions the use of methylene blue in 1-grain capsules.

Veronal and trional in combination in a dosage of 0.15. gram ( $2\frac{1}{2}$  gr.) each, if given every two to four hours, calm the pain, and induce restful sleep when the attack is over. Lockwood also advocated larger doses of antipyrin—1 gram (15 gr.)—given by the bowel every four to six hours.

Cannabis indica and belladonna to control hypersecretion, and the bromids, chloroform, cocain, and alkaloids of opium to control pain have been tried with success by Friedenwald and Leitz. They also recommend the external applications of sprays of ether or ice, the  $x$ -ray, radium applications to the epigastrium, and the use of the galvanic current with a milliamperage of 10 to 15, with the negative pole applied to the abdomen, and the positive pole over the dorsal vertebræ.

Finally, in most cases one must have recourse to hypodermics of morphin, which should be used cautiously and sparingly on account of the danger of habit formation, to which such sufferers are extremely susceptible; and when given it should always be under the personal supervision of a physician.

The injection of adrenalin chlorid in 0.5 mil (8 *m*) of

1:1000 solution should be tried, since it is safe, and claimed to be efficient in the relief of pain, and is, therefore, worth a trial before morphin. It is well to be familiar with the range of blood-pressure in the individual case, and to reserve the use of adrenalin for such cases as exhibit a hypertension during the attack. Likewise the vasodilators (sodium nitrite, amyl nitrite, etc.) may be tried, as recommended by Barker and Raymond, in those cases of hypertension in which the pain is contributed to by arteriospasm.

*Intercurrent Treatment Between Attacks.* The patient should be brought to, and made to maintain, the highest level of health. His diet should be simple, and made compatible with his gastro-intestinal digestive chemistry and tolerance, but should be of a high caloric value, containing an abundance of fats and oils, in the form of cream, butter, olive oil, bone-marrow, codliver oil, malt, cheese, etc., with a sufficient amount of proteins and carbohydrates. It has been truly said that a tabetic who is gaining in weight is doing well. The meals should be frequent and small. If during the acute attack gastric dilatation has occurred, during convalescence and for some time thereafter the patient should lie down on his right side for an hour after each principal meal, with the foot of the bed or couch elevated 6 or 8 inches (15.2 to 20.3 cm).

Any state of anemia should be combated, probably best by intramuscular injections of cacodylate of soda and citrate of iron in a dosage of 0.06 gram (1 gr.) each, and glycerophosphate of soda 0.15 gram (2½ gr.). These may be given every other day, or Fowler's or Donovan's solution may be prescribed. All act as useful tonics.

Cardiac or vasomotor irregularities should be corrected. When due to an associated splanchnoptosia, proper abdominal support should be provided, the Curtis abdominal pad for men, and the Gossard corset for women, being suitable for this purpose.

It is important to mention the necessity of keeping the bowels open. Constipation is the rule, and in some cases can be a real menace to health, and actually provoke a gastric crisis, allowing the accumulation and absorption of the toxic Beta ethylamins, to which Holmes attaches great importance, in initiating an attack.

The dietary should, therefore, include stewed fruits, honey, treacle, figs, dates, and whole-wheat bread. Liquid petrolatum is most efficient. The agar preparations, impregnated with various laxatives, such as cascara, are useful. Attention to habit formation in attending to the bowel movements should be insisted upon. Likewise the use of proper abdominal exercises.

When there are special indications for treatment to correct gastric symptoms, whether organic or functional, these should be carried out. For example, in one case the daily morning removal of the fasting gastric residuum has done more than anything else to keep this patient in comfort, to forestall further critical attacks, and to build him up to a point where a pyloroplasty or gastrojejunostomy for pyloric ulcer can be performed with minimum risk. So, too, in a second case an operation for the correction of an organic duodenal lesion was carried out during the intercurrent period, after the patient had been properly built up, and the postoperative result has been most favorable.

During this intercurrent period attempts should be made to improve the local nutrition of the spinal cord. Counter-irritation in the form of dry cuppings, the cautery, galvanism, vibratory massage, etc., may be applied over the thoracic vertebræ. So, too, one may find help in the use of the high-pressure hose of alternating hot and cold douches to the spine. Exercises designed to stretch the spinal cord have been devised—the simplest, and perhaps as effective as any, being for the patient to lie flat on his back, and, with heels on floor, raise his body to a sitting position, and conversely to keep his head on the floor and raise the heels, so that the feet are at as acute an angle to the trunk as possible. Finally, the patient should *rest*, physically and mentally, and as far as possible out-of-doors. Tabetics do best in summer, and in a dry warm climate.

Anti-luetic therapy should be pushed to physiological tolerance. Upon this depends our greatest hope of arresting the progress of the disease, and in this way preventing further attacks. The various forms of mercury with potassium iodid are effective in breaking down the spirochetal invasion, and the arsenical preparations, salvarsan and its allies, by intra-



muscular, intravenous, and intraspinal injection, are invaluable in this endeavor. These remedies should be given in courses, alternating with periods of mercury and the iodids, and with salvarsan.

All cases should be closely followed and treated until, and for some time after, all active symptoms and findings have been eliminated. Certainly intensive treatment should be persisted in until the spinal fluid cell-count has been restored to normal, until the Wassermann reaction has become negative, and, if possible, until the globulin excess, which is the last to clear up, has been reduced to normal.

The operative treatment has already been discussed, and need not be recounted here, except to warn once more against too hasty recourse to surgery, until the attacks are unbearable, and all other measures have proved failures.

## DISEASES OF THE INTESTINES.

### ACUTE ENTERITIS.

This affection is all too often confounded with diarrhea, since the cause of each is largely the same, the presence of large watery dejections being the cardinal symptom of acute enteritis. There may be fifteen, twenty, or more stools in the course of twenty-four hours, the abundant liquid resulting either from diminished absorption or from serous exudation from the intestinal wall. The evacuations are homogeneous, or contain particles of undigested food. The color of the stool is dependent upon the quantity of the bile or blood present. The absence of bile gives rise to the clay-colored, feculent stool, while the escape of large quantities of bile into the intestinal canal produces a grass-green evacuation. As the movements diminish in number, the color becomes yellow or yellowish-brown. Blood is rarely present in the acute variety, and the same applies to the presence of mucus. The discharges may be of a serous nature, combined with a small quantity of undigested food, or have a semi-solid consistency. Microscopically, they consist of large masses of epithelium, many varieties of fungi, mucous leucocytes, calcium phosphate, oxalates, and shreds of undigested food (starch-gran-

ules, fat, vegetable, and muscular fibers). The stools are alkaline in reaction.

The passage of the large watery discharges produces a feeling of prostration, and the patient complains of pronounced languor and persistent headache. The temperature mounts to 103° F. (39.4° C.) or higher. There may, or may not be loss of appetite, nausea, and vomiting. Abdominal pain, of a spasmodic character, usually associated with rumbling borborygmus, is characteristic of the attack. The pain is usually referred to the lower abdomen, but it may be located in the colon, or follow the course of the sigmoid flexure; the presence of the inflammatory process in the rectum occasions painful tenesmus.

#### TREATMENT.

If the attack be caused by some indiscretion in diet, a mild purgative, followed by regulation of diet, is usually all that is required. A full dose of castor oil, or the exhibition of calomel in broken doses, is generally most efficient. Albuminous food in liquid form, such as skimmed milk or broths, and the employment of various milk modifications, such as junket, peptonized milk, milk and seltzer, lightly boiled or poached eggs, and oysters, all are to be recommended. When the chief seat of the disease is in the large intestine, easily digested starches and certain green vegetables are allowable; these include sago, lettuce, watercress, and arrowroot. The tendency to drink water constantly should be restrained, as tending to increase the diarrhea, and in its place may be substituted oatmeal water, cold, weak, unsweetened tea, barley water, brandy and soda, or iced champagne. Rest in bed is especially beneficial, in that it tends to keep the abdomen warm and to mitigate the pain. The use of the hot water bottle, the employment of spice plasters, mustard poultices, and the judicious application of any of the various rubefacients often distinctly relieves the pain. A flannel bandage should be worn day and night. When the chief tenderness is limited to the right iliac fossa, a simple enema, given slowly, will exert a marked sedative influence upon the colon, and offers a far more rational mode of treatment than would the administration of a cathartic. Chief reliance is to be placed on intestinal antiseptics and astringents. Among the former, salol stands preëminent.

Naphthol and strontium salicylate are both antiseptic and carminative. Any of these may be prescribed, singly or together, and in combination with carbolic acid and one of the bismuth salts. In certain cases the use of opium acts most happily, and the same applies to argentic nitrate and the extract of hyoscyamus. In those instances where the intestinal juices have been diminished or modified by the pathologic process, a combination of pancreatin and sodium bicarbonate offers an effective treatment.

In the employment of astringents, in an effort to check the diarrheal discharges, many a physician defeats the very object that he is seeking to attain, and he too often wonders why failure is the reward of his endeavor. All the vegetable astringents are irritants. The discharges from the bowels are provoked by an inflammation, and the effort of the physician should be to correct the condition that provokes the diarrhea, rather than to cure the diarrhea itself. Lead acetate is more sedative and less irritant than the vegetable astringents. In the latter stages of the disease the condition may be one of intestinal relaxation, when astringents will be demanded; these, of course, include such medicaments as sulphuric acid, chalk mixture, catechu, and kino. For the distressing flatulence, the alkaline carbonates, or spirits of ammonia, and a carminative may be used as a corrective. If the colicky pain be severe,  $\frac{1}{8}$  or  $\frac{1}{6}$  of a grain (0.00810 or 0.01080 Gm.) of morphin hypodermically may be given, in addition to the measures above outlined.

### CHRONIC ENTERITIS.

Chronic enteritis is anatomically divided into the chronic catarrhal form, the pseudomembranous (in which the copious mucoid secretion takes on the form of membrane or casts), and the ulcerative form. In chronic enteritis, diarrhea and constipation often alternate. Usually there are about six stools during the course of the twenty-four hours; pain may or may not be a symptom, but the patient complains of a feeling of weight and discomfort in the abdomen.

The evacuations contain much mucus, often with little fecal matter, or the feces and mucus may be intimately mixed. There may be distention of the abdomen, with tympany and

the occurrence of borborygmus. Tenderness on palpation may or may not be a symptom. The patient may appear well nourished or be emaciated. The victim of this affection is nervous, irritable, dissatisfied, easily fatigued, and morose.

#### TREATMENT.

The hygienic treatment of this troublesome affection is all-important. The body must at all times be uniformly warm and dry. Silk or all-woollen undergarments must be constantly worn. Even the ankles must be protected against changes in temperature, and with the approach of cooler weather the wearing of the house slipper is to be guarded against.

The dietetic management of the case is of paramount importance. In severe cases it is often advisable to put the patient upon a milk diet for several weeks. As a rule, fatty or saccharine substances are interdicted. Slow eating and thorough mastication are imperative adjuncts in the dietetic régime. The menu should include scraped beef, lean meats, and bread. If this diet is too sparse, there may be added pancreatinised food; and baked potatoes and other farinaceous foods may be treated with diastase or malt extract, although as a rule starchy foods are strongly contraindicated. Generally, no vegetables should be allowed; well-boiled rice in some cases acts as a happy substitute. Coffee is to be forbidden; tea may be used in moderate amounts. Ice-cold drinks are injurious, and among the foods to be avoided may be cited: very rich milk, green vegetables, raw acid fruits, nuts, all fat dishes, lobsters, crabs, shrimps, pork, veal, and all sweets. The best form of alcoholic stimulant is claret, sherry, or brandy, diluted three or four times its volume with plain water, Vichy, or Appolinaris. The use of various mineral waters is urged by many observers; and residence for some weeks at one of the alkaline mineral springs finds many advocates.

The amount of exercise required must be regulated by each particular case. In those cases where there is pronounced exhaustion, rest in bed is to be enjoined; and the application of Swedish movements is most essential. In other cases carefully graded exercises are to be advised.

The medicinal treatment consists of daily irrigation of the

bowel with some antiseptic solution, such as salicylic acid, 5 grains (0.32 Gm.) to the ounce; boric acid, 10 or 20 grains to the ounce (0.65 or 1.3 Gm. to 30 mls); creolin, 5 grains to the ounce, (0.32 Gm. to 30 mls); and for its alterative action, the nitrate of silver is often the agent *par excellence*, in the proportion of 4 or 5 grains (0.26 or 0.32 Gm.) of the nitrate of silver to each pint (473 mls) of water to be injected. The fluid should have a temperature of 90° or 95° F. (32.2° or 35° C.), be introduced slowly, and at least one quart (1 l.) of the solution introduced at each sitting. These irrigations are much more to be depended upon than the employment of astringent remedies, because the arrest of the diarrhea by means of astringents is only temporary, and is almost always succeeded by an increase in severity of the original condition rather than by its betterment. Among the more valuable internal remedies may be mentioned the nitrate of silver, lead acetate, bismuth, especially in combination with carbolic acid, or turpentine. It should always be borne in mind that all agents intended to influence the intestinal mucous membrane should be exhibited an hour or two following a meal, corresponding to the time when the stomach contents are passing into the small intestine. In prescribing certain drugs, such as the nitrate of silver, the fact must not be lost sight of that destructive changes in the presence of the gastric juice makes it incumbent upon the physician to instruct the pharmacist to enclose such a pill in a double capsule, so that the medication may exert its full influence upon the inflamed surface of the bowel. This affection is difficult of treatment, so that remedial measures need to be energetically and patiently carried over long periods of time.

### ENTERITIS IN INFANTS.

This scourge of our large cities during the hot season is variously designated summer diarrhea, enterocolitis, diarrhea and enteritis, febrile diarrhea, and inflammatory diarrhea. It is always associated in the lay mind with the "second summer," although isolated cases are not rare during the winter season. Hot, damp, weather is especially conducive to the condition, and of all the months August offers the greatest



mortality, because during this month the high daily temperature is maintained throughout the night.

The greatest number of victims is found between the ages of six months and eighteen months. The invasion of the malady depends upon the sympathetic irritation of the alimentary canal; in association with the eruption of the teeth, the increased tendency to inflammation, engendered by the rapid development that the intestinal follicles and glands are simultaneously undergoing, and the fact that weaning is often practised during this critical season. In brief, the hot season, contaminated city atmosphere, bad food, improper housing, insufficient nutrition, unsuited articles of diet, and excesses of farinaceous food, all are potent factors in the production of the condition. Breast-fed babies are less prone to enteric affections, but they also may fall victims from too frequent or continuous feeding, or from abnormalities in the mother's milk.

Twenty-four or forty-eight hours before the attack the child is restless and fretful, his sleep is broken, he looks pale, and the mother tells the doctor that the child's head "feels feverish." The baby will not take the bottle, or if it does, sour eructations are almost certain to follow, and at the same time the bowel movements are frequent and soft in consistency. Obstinate vomiting now sets in, and this is followed by a large number of loose stools, acid in reaction, and passing from yellow to a grass-green in color. The diaper may appear "slimy," or much mucus stained with blood is passed. So intense are the symptoms at times that the tenesmus occasioned may be associated with slight prolapse of the rectum.

The tongue is dry and red at the tip and edges, there is no appetite, and thirst is increased. For the first few days there is moderately high fever, and later the pyrexia is remittent; the pulse is weak, and runs as high as from 120 to 140 per minute; the urine is scanty, high-colored, and passed at long intervals.

As the diarrhea continues, the face becomes pale, the eyes sunken, the muscles flabby, and there is intense prostration. The decreased amount of the urine may herald the fatal termination, with uremic poisoning as the cause of death.

### TREATMENT.

The prophylaxis is the avoidance of the dangers of summer diarrhea by taking the child to the country, mountains, or sea shore, where the air is cooler, and uncontaminated, and where pure and clean milk can be obtained.

Among the poorer classes the child should be taken to the public parks, also on a morning and evening trip on a river boat, be given cool baths, and be cleanly clad. The room and the bed should be scrupulously clean. Good ventilation is a prime essential.

The child should not be fondled or carried; it should be kept in bed or wheeled around in a coach. Light woolen clothing should be next the child's skin; otherwise the apparel should be of the lightest texture. Twice or thrice daily a bath of 80° F. (26.7° C.) is an excellent measure; with marked prostration, the warm bath is to be urged.

Overfeeding at the breast is to be guarded against, and the same applies to the bottle-fed baby. The high fever and loss of fluid in the discharges produces a marked thirst, and it is important to see that the milk secured for the little one is absolutely fresh, and from a responsible dealer. The milk should be administered from an absolutely clean bottle, and in the intervals the rubber nipple always should be turned inside out, cleansed of all curds and detritus, and thrown in a vessel of water in which a small quantity of borax or sodium bicarbonate has been previously dissolved. When the milk or one of its modifications is vomited, or passes undigested from the bowels, a whey mixture may be employed, beef juice may be tried, or flour ball; or, if none of these seems to agree with the child, all food should be withdrawn for from twenty-four to forty-eight hours, and the infant placed upon barley water. Cool sterile water and particles of ice may be used to relieve the intense thirst.

The drug treatment consists in emptying the bowels by means of castor oil; in great irritability of the stomach, an enema should take its place. For a child of six months, a pint (473 mls) of water at a temperature of 65° or 70° F. (18.3° or 21.1° C.) is to be employed, and the injection slowly given. Calomel and the salicylate of sodium are both antisept-

tic, and are capable of great good. The calomel can be given in the usual small broken doses; the salicylate in doses of a grain or two (0.065 to 0.130 Gm.), dissolved in peppermint or cinnamon water. Counterirritation to the abdomen, or application of different forms of heat, or the spice plaster, are all beneficial and comforting. When prostration sets in, stimulants are demanded, depending in quantity and frequency upon the age and the condition of the child.

### CHOLERA INFANTUM.

The severest and gravest form of acute enteritis in infants is known as cholera infantum. Here the vomiting becomes excessive, and the watery stools may number thirty or more in the twenty-four hours. Painful cramps in the muscles of the extremities may take place, and a condition of collapse rapidly supervenes. The superficial temperature is often subnormal, but the rectal temperature shows the presence of fever, and there may be hyperpyrexia, anteceding death.

The stomach becomes irritable, refusing everything; even ice is rejected. The child drinks with avidity at every possible chance, piteously looking at the receding empty glass—a mute appeal for a cooling substance to quench the consuming thirst.

A little later the baby lies still and apathetic; the face is drawn, the skin is clammy, the pulse is small and rapid; and when death supervenes not uncommonly it is preceded by a convulsion; or the child first passes into a prolonged coma that gradually fades into an endless sleep. The prognosis always is grave. Death often occurs within forty-eight hours after the invasion of the disease. In cases of recovery, convalescence is much protracted.

### TREATMENT.

What has been said of the treatment of acute enteritis in infants applies with equal force in treating cases of cholera infantum (*v.s.*). The large watery evacuations are so exhausting to the child that it becomes at once imperative to check these discharges, and to maintain the bodily strength by food and drink. In spite of the irritability of the stomach,

every effort must be made to give food in small quantities, and at short intervals. To check the diarrhea, opium and astringents need be exhibited. Stimulants are demanded very early in the affection to prevent the occurrence of prostration. Whisky or cognac, 5 or 10 drops (0.30 or 0.60 mls) in a dram (3.75 mls) of limewater, may be given every thirty minutes to a child six months old. When collapse occurs, the amount of stimulant must necessarily be increased, and given at more frequent intervals. In conjunction with alcoholic stimulants, a diffusible cardiac stimulant as the carbonate of ammonia is especially useful.

The temperature must be maintained by hot water bottles, and the child kept in a lying posture, and disturbed as little as possible. The presence of cerebral symptoms is a contra-indication to the use of opium. For other details of treatment, including the hygienic management, etc., the reader is referred to Acute Enteritis in Infants. (See p. 841.)

### ACUTE COLITIS.

This is one of the special forms of enteric catarrh, each variety manifesting certain symptoms, according to the particular part of the bowel affected, and these are variously designated: duodenitis, localized catarrh of the jejunum and the ileum, and proctitis. The joint appearance of abdominal pain and loose dejections is almost diagnostic of acute colitis. There is tenderness on palpation over the region of the colon; the stools contain blood, and often large quantities of mucus, the latter not being mixed with the fecal mass, as in catarrhal conditions of the small intestine. The patient appears pale and emaciated; weakness and sallowness of the skin are often observed.

The *treatment* is that of acute enteritis (*v.s.*).

### CHRONIC MUCOUS COLITIS.

The distinction made between mucous and membranous colitis is one that cannot well be maintained. The peculiar symptom-complex variously described as membranous colitis, membranous enteritis, pseudomembranous enteritis, and fibrous enteritis, is primarily a disease of the female sex.

From 80 to 90 per cent. of the cases are found in women. These women all exhibit well-marked neurotic or hysterical symptoms; and even the small proportion of males affected evidence the same nervous phenomena. In 1871, Dr. J. M. Da Costa, of Philadelphia, wrote a most elaborate treatise on the subject, and, although almost half a century has since elapsed, nothing of an important character has been added to the knowledge contained in that masterful essay.

Originally Da Costa maintained that mucous colitis was no colitis at all, but a secretory neurosis, attended with an excessive secretion of mucus in the colon, and it has been frequently suggested since that time that all the synonyms mentioned at the opening of this article be dropped, and in their place the term membranous or mucous colic be substituted.

The affection is characterized by the passage of a greyish-white translucent substance, preceded by a colicky pain. Sometimes these masses appear as shreds, or lumps, or balls, or as membrane conforming to the shape and caliber of the intestinal canal. The amount of this substance may be large or small, and the feces, as a rule, are rarely passed with this material. When allowed to float upon the surface of water these masses are rolled up, and produce a lumpy appearance, while chemically the substance is found to be mucin, although at first it was incorrectly designated fibrin; hence the erroneous term, fibrous enteritis. It is worthy of note that leucocytes are found in extremely small numbers in this newly formed substance; hence there is no suggestion in the study of the subject to suspect inflammation or suppuration.

With the discharge of these mucoid masses the colic is at once relieved. There may be intervals of weeks, or even months, between these attacks, the patient appearing perfectly well in the interval. The length of the attack may be hours, days, weeks, or months. The pain is described as cutting; burning, shooting, or stabbing. The pain may be felt at the sigmoid flexure or cecum, and radiates most often toward the umbilicus and epigastrium, or at times down the thighs, especially on the left side. Nausea is often a symptom; but there is no vomiting and no fever. Enteroptosis and gastric hyperacidity are often concomitant symptoms. Pathologically, no anatomic lesion has up to the present time been found.



## TREATMENT.

The therapeusis of this peculiar malady is difficult of description, because of the neurotic or hysterical element too often forming the groundwork of the symptom-complex. The patient's confidence must be won at the outset. The diet should be as liberal as possible, in order to sustain the bodily vigor and to counteract constipation. Exercise in the open air and hydrotherapeutics are to be encouraged. Lavage of the bowels, by means of high injections, thus causing colonic flushings, are to be highly recommended. Astringent injections, especially of the nitrate of silver, are of decided benefit. These enemata should be employed at least thrice weekly, and may be varied by the addition of borax or common salt in the proportion of 2 or 4 per cent. up to saturation. Two quarts (2 l.) of solution should be used at each sitting, the water having a temperature of 105° F. (40.5° C.). It should be remarked, however, that not a few clinicians mention lavage and injections in these classes of cases merely to condemn these procedures as productive of an increased secretion of mucus in the colon.

The abdominal bandage should be worn day and night, and daily bathing is an invaluable hygienic measure not to be overlooked. In the dietary, oatmeal is forbidden; potatoes, beets, and other vegetables that grow under the ground are prohibited; spinach, young peas, or lima beans can be sparingly eaten. Rice, cheese, and milk foods are permitted. Toast bread and pulled bread are far more preferable than fresh bread. Tea is to be chosen rather than coffee or chocolate. Alcohol may be used sparingly, but malt liquors are especially injurious. During the paroxysms the patient should be kept quiet in bed; counterirritation along the course of the colon may be practised in suitable cases, and the administration of castor oil until its effects are manifest should be a routine practice, at the same time using the injections as just described. Diarrhea is to be controlled by some mild astringent, of which dilute sulphuric acid is the drug *par excellence*. In the event of constipation, no hesitation should be felt in the use of laxatives, which should be varied from time to time. Among the more efficient ones may be cited: cascara, an oc-

casional dose of calomel, a combination of the vegetable cathartics, sodium phosphate, and the A.S.B. pill. The salines in uncomplicated cases, in which there is no catarrh of the bowels, are not to be recommended. Opium is to be used only to relieve pain and tenesmus, and other measures should be first employed before resorting to this drug, which really has no place in the treatment of mucous colitis.

### ULCERATIVE COLITIS.

Ulcerative colitis is often associated with chronic intestinal catarrh, and is a not uncommon complaint. As a rule, the ulcers are quite extensive, and tend to denude the greater portion of the mucous membrane. Two varieties are recognized: the catarrhal, extending from the surface downward; the other, the follicular, proceeding from an abscess of the lymph follicle in the wall of the bowel.

Ulcers of the intestine give rise to few characteristic symptoms. Indeed, diarrhea may be the only symptom present; and when the ulcers are large and numerous, the course of the affection is that of acute or chronic enteritis. Pain is not a dependable symptom, but when present it is of a colicky, griping nature. When sharply localized, and markedly sensitive in that particular region, extension of the morbid process to the peritoneum is strongly suggested. The presence of pus, blood, or shreds in the dejections would be strongly confirmatory of the existence of ulcer, but any of these is rarely to be found. When the amount of pus discharged is at all appreciable, the presence of abscess and not ulcer is to be surmised.

The general health suffers little from the existence of these ulcers. But perforation of the ulcers, depending upon their location, may lead to a general peritonitis. Stricture of the bowel or embolism of the portal vein may follow ulceration.

### TREATMENT.

The treatment of ulcers in the upper bowel is practically the treatment of chronic enteritis. In ulceration of the large intestine the treatment is virtually that of chronic enteritis, plus local medication. Intestinal antiseptics, such as salol,

are especially valuable, as they remedy complicating conditions in the small intestine. Bismuth is perhaps the only drug taken by the mouth which reaches the large intestine; it must always be prescribed in large dosage.

Greatest dependence, however, is to be placed upon intestinal irrigation. Twice or thrice weekly, an injection of 2 quarts (2 l.) of water, containing 30, 40, or 50 grains (1.95, 2.60, or 3.25 Gms.) of nitrate of silver, is often productive of most excellent results; and in the intervals, once or twice weekly, the bowels are to be irrigated with a saturated solution of borax.

In the treatment of ulcerative colitis during the acute stage, much of the success to be obtained consists in the careful regulation of the diet, confining the patient's dietary to liquids and semisolids, and maintaining the bowels in a soluble condition.

### APPENDICITIS.

The term appendicitis was first proposed by the late Professor Reginald H. Fitz, of Harvard University, to designate that important primary disease of the right iliac fossa that, among other appellations, had been variously called iliac abscess, iliac phlegmon, perityphlitis, and paratyphlitis. In his elaborate studies, he emphasized the fact that the above-named affections had so many points in common with perforation of the vermiform appendix: "that, for all practical purposes, typhlitis, perityphlitis, typhlitic tumor, and perityphlitic abscess meant inflammation of the vermiform appendix; that the chief danger of this affection is perforation; that perforation in the great majority of cases produces a circumscribed suppurative peritonitis, tending to become generalized."

The great frequency of appendicitis, finds a ready etiology both in congenital and acquired conditions. Thus, in the newborn babe, the appendix may be unusually long, or it may occupy an abnormal position, or there may be something anomalous in the development of its mesentery, which abnormalities all tend to favor the accumulation of matter within the canal. In the acquired variety, the existence of adhesions dependent upon inflammation of the appendix or other part of

the general abdominal cavity, may bind down the appendix and prevent it from expelling its contents. Digestive disturbances may be a cause, and the occurrence of influenza as favoring the development of the affection finds not a few advocates. Fecal concretions, foreign bodies, strains, jars, and traumatism all are factors of etiologic importance. Often, however, the disease occurs without apparent cause. It is commoner among males than females, and statistics record its presence in a child of eighteen months; and then again in a man of eighty. It is most frequently met with between the ages of ten and thirty; and out of three hundred *post mortems*, the appendix has been found to be diseased in one hundred and ten instances.

The appendix may be affected and the attack so latent, as either to produce no symptoms, or the inconvenience be so slight as to be inconsequential to the patient. Ordinarily, however, appendicitis may be classified as acute and chronic.

The acute variety is characterized by abdominal pain, slight or severe, in association with a chill or some chilliness. Possibly for a day or two the patient has felt somewhat indisposed with slight undefinable symptoms, or, as often is the case, the sufferer was enjoying, apparently, the best of health.

The pains are at first diffused over the abdomen, probably because the superior mesenteric plexus of the sympathetic supplies both the appendix and a large portion of the intestines; a little later the discomfort manifests itself at the umbilicus, where the intensity of pain, following the physiologic law, is felt at the nearest great nerve center, which in these cases are the great abdominal sympathetic ganglia situated in the umbilical region. The pain, at first colicky, is localized a few hours later in the right iliac fossa, when a neuritis has developed of sufficient grade to cause tenderness on pressure.

This point of pain upon pressure, commonly designated "McBurney's point," is near the outer edge of the right rectus muscle, on a line between the navel and the anterior superior spine of the ilium. Depending upon the topographic position of the appendix, the point of greatest tenderness may be found elsewhere in the right iliac fossa or even in the left iliac fossa, the groin, and either the umbilical or the lumbar region.

Vomiting commonly follows, with little relation to the gastric conditions, and is ordinarily reflex and due to reversed peristalsis. The material ejected is that which the stomach contains; a little later it consists of mucus, sometimes bile-stained; but the stercoraceous vomiting of hernia is not encountered.

Moderate fever,  $99.5^{\circ}$  to  $101^{\circ}$  F. ( $37.2^{\circ}$  to  $38.3^{\circ}$  C.) with a corresponding increase of pulse-rate are usually present.

There is slight rigidity of the right rectus abdominalis muscle, and later of the musculature over the right iliac fossa. Respiration is but little affected. There is anorexia, and the vomiting before mentioned is a symptom of the onset of the malady, but later disappears unless general peritonitis is present. Constipation is the rule, although diarrhea sometimes precedes the attack, and may be a late symptom in protracted cases.

So long as the disease is limited to the appendix, the swelling is not well made out; in those cases where the appendix lies posterior to the cecum, the swelling is impossible of demonstration.

### TREATMENT.

Although a discussion of surgical measures would be irrelevant in a work such as this, we need recognize three conditions when surgical interference and not medical treatment becomes a *sine quâ non*.

1. *In all cases where the initial symptoms, the pain, tenderness and tympany are excessive, with marked acceleration of the pulse and temperature, indicating in all probability the occurrence of an acute perforating appendicitis.*

2. *In cases of apparently mild appendicitis, where after seventy-two hours of treatment, there is no amelioration of the symptoms. Except in those cases where fecal masses have been discovered, at the onset of the attack, at the position of the head of the colon, especially if there has been tenderness along the course of the colon and not in the immediate vicinity of the appendix.*

3. *An immediate operation is called for in cases showing the symptoms of perforation—that is, collapse; also in cases where the symptoms point to development of suppuration—the exquisite*



*tenderness, elevation of temperature and acceleration of the pulse-rate, being the ominous symptoms indicating the oncoming of a general peritonitis.*

When an appendicitis attack has progressed several days before the doctor has been called in—and in fact, often at the outset of a case—it is the practice of many careful physicians to ask for the opinion of a skilled surgeon. Let it be remembered, that about the third or fourth day of the affection, abscess formation is likely to occur, when the inflammation tends to circumscribe itself by throwing out lymph and forming adhesions, not very strong—a critical time, in which “it is too late for an early operation and too early for a safe late operation.”

These are the delicate questions that require nicety of judgment on the part of the attending physician. Having determined that the case is one for medical treatment, the patient should be kept in bed in a well-ventilated room, and absolute quiet be enjoined. The diet should be liquid and sustaining. It should consist of foods that will be thoroughly absorbed, leaving as little residue as possible to irritate the large intestine and provoke peristalsis. The diet should be made up of nutritive broths, beaten eggs, pancreatized milk, whey, milk well diluted with seltzer acid water, or lime water, and butter-milk.

At the onset of the disease, especially if a “sausage-like” tumor be palpable, intestinal irrigation, with a view to removing the fecal accumulation, is to be assiduously practiced. The use of saline laxatives, until the bowels have been thoroughly emptied should be a routine practice. It is the custom of very many careful clinicians to administer calomel during the course of the disease, providing that there are no symptoms of gangrene, perforation or serious septic infection. If the physician suspects ulceration or gangrene of the appendix then purgatives should *only* be resorted to to cleanse the bowels, if fecal accumulations can be demonstrated. It is a matter of clinical experience that the presence of fecal matter in the colon greatly increases the danger from operation upon the appendix, a fact that must never be lost sight of. As has been just remarked, in all cases of appendicitis high injections are always a valuable measure; and these are absolutely to be de-

pended upon in ulceration or perforation of the bowel, the use of any laxative or purgative agent by the mouth, in the presence of these complications, being absolutely contraindicated.

Local applications consist of the use of heat, cold, leeches, and blisters. The suspended ice-bag is an excellent means of relieving pain; but in its place cloths wet in ice-water may be applied and changed every few minutes. If warmth is more agreeable to the patient, the hot water bag may quite happily be substituted for the ice-cold application. Leeches are very effective, and there is no good ground for any antagonism concerning their employment. The same does not hold good of blisters. The latter are extremely discomforting and painful to the sufferer; their effect upon the spread of the inflammation is to be regarded as negligible, and the raw surface that they occasion is a handicap to any surgical interference.

There exists today some differences of opinion concerning the use of opium. The consensus of opinion, however, does not favor the use of the drug, for it checks secretion, interferes with peristalsis, masks the abdominal pain, and leaves the surgeon hopelessly wandering in the darkness, as to whether interference should be adopted or not. Unless, therefore, opium is called for by the presence of excessive and unbearable pain it is best to avoid it, and when demanded it should be given in the form of hypodermic injections of morphin, in doses of from  $\frac{1}{8}$  to  $\frac{1}{4}$  of a grain (0.00540 to 0.01620 Gm.). When operation has been decided on, morphin may be given, however, without hesitancy. Mild counterirritation, such as the use of mustard paste or plaster, is practised by some physicians, in the hope of offering some relief from the constant gnawing pain, but even these applications are prone to produce induration of the integument and the underlying tissues. Sleeplessness from pain is to be met with morphin; but insomnia which may proceed in the wake of the disease is to be treated with one of the synthetics, such as trional or veronal. Chloral in these cases is a valuable hypnotic.

As the patient convalesces, he should not be permitted to leave his bed for several days after the disappearance of all symptoms. During this period, the diet must be carefully regulated and the bowels maintained free and soft.

### CONSTIPATION.

The definition of habitual constipation might well answer for synonyms of the complaint itself: Chronic fecal retention; habitual infrequency of bowel movements; irregularity, insufficiency or difficulty of the evacuation of the bowels. The onward passage of the fecal current is dependent upon peristalsis. It requires four hours for the bowel contents to pass through the small intestine, and from twelve to twenty hours to pass from the cecum to the anus. In the vast majority of cases constipation is due to lack of peristaltic and expulsive power, and also a deficiency of hepatic and intestinal secretions. The causes of constipation may be conveniently grouped into three great classes:

(A) (1) *General* causes: These include persons of a nervous temperament, common among women, especially of a brunette type, and familiarly known among the laity as "torpor of the liver" or "sluggish bowels." A sedentary life, develops the "constipated habit," and this also applies to those who, from an innate sense of modesty, either do not permit the bowels to move in the retiring rooms of public places or who hurry the act, because of the ill-kept, unsanitary, and often semi-public character of these closets, all of which tend to lessen the sensibility of the rectum to the exciting action of the contained fecal accumulations.

(2) General bodily weakness and disease. In this category is included: Nervous affections—neurasthenia, hysteria, diseases of the brain and the cord, acute fevers, disorders of the liver, habitual use of purgatives, acquired degeneration of the muscular coat of the bowels, as in chronic enteritis and chronic peritonitis.

Abundant diuresis and diaphoresis, which abstracts large quantities of fluid from the system, may engender constipation.

(B) *Local* causes: These include atony of the abdominal muscles, as occurs from obesity, and repeated pregnancies; atony of the bowel caused by pressure from tumors; narrowing of the lumen of the bowel by growths within the intestine or by pressure from without, and from functional intestinal neuroses.

(C) *Dietary* causes: These include insufficient food, be-

cause the small bulk of aliment fails to excite peristalsis, and there is an accumulation of waste products as a consequence; food that is too rich or too highly concentrated is often completely absorbed, leaving insufficient residue of waste matter to provoke peristalsis; astringent food and drink, by checking mucous and other secretions, necessarily increase friction within the intestinal tube; indigestible food may be imperfectly acted upon by peristalsis and incompletely mingled with the digestive juices, or fermentation may develop and the production of substances be formed which interfere with absorption and peristalsis; irregularities in diet, or in the intervals of taking food, irregular mastication, etc., not infrequently find a counterpart in the irregularity of defecation or in its partial suppression; insufficient fluid is a frequent cause of constipation.

#### TREATMENT.

This comprises the hygienic, dietary, remedial, and mechanical measures. The *hygienic régime* includes the habit of defecation at a particular time each day (there should be at least one bowel movement in the course of the twenty-four hours). As sedentary habits are not infrequently the causative factor, systematic graded exercise, whether in the open, gymnasium, or at home, must be insisted upon. Walking to or from the place of business is applicable to every busy worker. A glass or two of cool water upon retiring, and again immediately upon arising, often exerts a markedly beneficial effect. At each call of nature, the bowels should be evacuated as completely as possible, and under no circumstance should the patient defer the act.

The *dietetic measures* are of incalculable importance. The principle of treatment is based upon supplying food which will provoke peristalsis, either by *bulk* or from its physical and chemical properties. The cellulose of certain starchy vegetables is difficult to digest, and is of little nutritive value, yielding a bulk of waste; among these are potatoes, corn, peas, and beans. Other vegetables leave a large residue to excite peristalsis. These include tomatoes, asparagus, spinach, cabbage, and celery.

The various cereals when *coarsely* ground contain a large

proportion of the external envelope of the grains, which mechanically irritates the intestines. Thus, the following articles tend to overcome constipation: rye, corn meal, Indian meal, oatmeal, Boston brown bread, whole-meal bread and wheaten grits. Again, the finest white flour favors constipation, because it contains so little of the innutritious part of the grain.

Molasses and honey smeared on bread are laxative. Brown sugar has a marked laxative action; white sugar possesses that power to a very slight degree; sugar of milk is absolutely inert. Among the fluids exerting a laxative action are: coffee, beer, cider, unfermented grape juice, olive oil, and cod-liver oil. The last two are especially useful, when there are evidences of failing nutrition associated with constipation.

Fruits possess a laxative action either because they contain indigestible seeds that mechanically irritate the intestines, or from some inherent property are capable of bringing about a chemical action. Many fruits act in both ways. Fruits containing seeds are: strawberries, blackberries, huckleberries, figs, and grapes. (Grapes are laxative when eaten in quantity and the seeds rejected.)

Fruits enjoying special laxative properties include: apples, oranges, peaches, cherries, prunes, and plums. Among the fruits combining both of the above advantages are: prunes, raisins, figs, and apples.

Where acid fermentation results from the ingestion of raw fruit, then stewed fruit becomes a useful and an agreeable addition to the dietary. The only objection that may be raised against partaking of stewed fruit is that so much sugar is required in its preparation as to disorder the stomach and provoke flatulency. Canned and preserved fruits are of little value. Bananas are constipating.

The patient should take many draughts of water during the course of the day. Water may be taken *with* meals; the statement offered that the liquid dilutes the gastric juice and thus interferes with gastric digestion has no basis in fact; *per contra*, the addition of water to the gastric juice does not materially dilute that powerful acid secretion, but rather increases its quantity for action upon various foods concerned in stomachic digestion.



Persons of a constipated tendency should avoid eggs, milk, sweets, fried foods, gravies, sauces, strong condiments, pickles, tea, and sour and red wines.

*The medical treatment* of constipation often becomes a necessary evil. It should always be regarded as a last resort. The broad rules to be followed in the use of drugs may be tabulated as follows:

1. Use laxative drugs only after other measures have failed.

2. Their use should be continued daily in small doses until the stool becomes soft.

3. Drugs should be changed or alternated so that the bowels do not become accustomed to any particular remedy, thus engendering constipation.

Laxative drugs include the salines and vegetable laxatives. The salines include, besides the various mineral waters, Rochelle, Epsom, Glauber's salt, and the citrate of magnesia. All salines should be given, very much diluted, the first thing in the morning on rising. This is imperative, because saline laxatives act upon the empty bowel and favor peristalsis by abstracting the watery elements from the intestinal blood-vessels. Among the many vegetable laxatives is mentioned cascara, compound licorice powder, the compound cathartic pill, U. S. P.; the aloin, belladonna, strychnin, cascara pill, senna, podophyllum, and rhubarb. Each of these drugs possesses certain virtues, and may or may not be adaptable to every case.

In atonic conditions of the intestines it is frequently advantageous to combine drugs that increase peristalsis. Thus, physostigma is a stimulant to peristalsis by augmenting muscular activity in the intestinal walls. Hyoscyamus and belladonna increase peristalsis by a depressing action on the peripheral ends of the inhibitory fibers of the splanchnics and by decreasing any tendency to spasm of the muscular coat of the bowel. Atropin can likewise be employed; while strychnin added to purgative pills avoids the depressing after-effects on the intestine. The rationale of ordering strychnin, hyoscyamus, and physostigma in combination with cathartics such as aloin or podophyllum, is well exemplified in many laxative pills. A combination of cathartics often relieves the harsh

action that results if one is given singly. This is well illustrated in the well-known compound cathartic pill, U. S. P., composed of colocynth, jalap, gamboge, and calomel, a combination which is very efficient as a purge, and unattended with much griping. Eserin acts as a stimulant to the muscular coat of the bowel, and is especially valuable in elderly persons and others whose intestinal muscular fibers are failing in power. The average dose is  $\frac{1}{30}$  of a grain (0.00216 Gm.).

Ordinarily, it is best to give vegetable laxatives at bedtime, because some hours are required for their action. In cases of obstinate constipation, good results are often attained by administering the vegetable laxatives after each meal. The use of liquid petroleum products have received a measure of approbation because of the lubricating action which they have upon the intestinal contents, in this way favoring peristalsis.

*Mechanical measures* include the use of enemata and suppositories. Their employment may be resorted to as an occasional substitute for laxatives. They act upon the rectum and the lower colon, but their use is limited since they soon obtund the sensibility of the rectum. When the action of the laxative medicine fails to assert itself, or if the stomach is unretentive, the use of a suppository or enema may be substituted. Massage, stimulating the abdominal muscles and peristalsis, may be practiced at regular periods during the day and at times corresponding to that of desired evacuations. The abdominal muscles and peristalsis are stimulated in this way. The rolling of a heavy ball or cannon ball along the course of the large intestine is often effective. In the obese and persons with pendulous abdomens, as is seen among women who have borne many children, the adjustment of a suitable belt or tight binder will not infrequently act as an important adjunct in treatment.

In cases of impacted feces much difficulty is often experienced in moving the bowels. All irritating measures should be avoided, especially drastic cathartics. Saline laxatives are well adapted for this purpose, as are combinations of vegetable drugs. Another useful laxative agent is the infusum sennæ compositum, U. S. P., 4 ounces (120 mils) at a dose. This is commonly designated "Black Draught," and is composed of senna, manna, and sulphate of magnesium. When

a hard inspissated fecal accumulation can be felt in or above the rectum, it may become necessary to remove it with the finger, aided by a spoon or other device employed for this purpose. Rectal injections forced beyond the seat of impaction may prove efficient in removing the hardened feces. These should be given hot to act as a solvent on the fecal accumulation. Only part of the mass should be removed at a time to prevent irritation.

## INTESTINAL OBSTRUCTION.

Intestinal obstruction, a condition which arises when the fecal current is impeded or prevented, may be incomplete or complete, acute or chronic. Acute obstruction is due to a *sudden* occlusion of the gut, while in chronic obstruction the narrowing of the lumen is *gradual*, but obstruction is likely to become acute at any time. The acute variety is most common in the small intestine; the chronic form, in the large gut. It is the chronic form that occurs most frequently in the aged.

Intestinal obstructions may be classified as follows:

*Strangulation.* This is the most common form, and is usually due to peritoneal adhesions. Constriction of a loop of intestine may be caused by the free end of a persistent Meckel's diverticulum attached to the abdominal wall.

Strangulation may also take place beneath an adherent appendix, a fallopian tube, or a portion of mesentery. This form of obstruction is identical with that occurring in hernia. Seventy per cent. of the cases of strangulation occur in males, and 40 per cent. in persons between the ages of 15 and 30.

*Volvulus or twists* in the intestine are most common at the sigmoid flexure of the colon. An unusually long or lax mesentery is a predisposing factor. A twist with a sharp bend in the bowel results in strangulation. Males between 40 and 60 are especially prone to the condition.

*Intussusception* is due to active peristalsis, and is the form of obstruction mostly always found in children. It occurs at the ileocecal valve, in the ileum or cecum, and occasionally in the rectum. It may be described as a telescoping of one section of the bowel into another. More than 50 per cent. of the subjects thus affected are under 10 years of age.

Among the other causes of obstruction are: intestinal stricture, either cicatricial or malignant, obstruction by tumors inside or outside of the bowels, by foreign bodies (gall-stones, enteroliths, intestinal calculi) and fecal accumulations. The latter are due to paresis or paralysis of the gut.

In acute obstruction of the bowels pain comes on suddenly, and is severe and colicky, accompanied by syncope, extreme prostration and shock. Vomiting is an early symptom. First the stomach contents are ejected, followed later by bilious vomiting, and finally the regurgitated matter is stercoraceous. The abdomen is tense, distended and tender. As shock passes off, the temperature becomes elevated; but in an unrelieved case there is a subnormal temperature, a cool, clammy skin, and a rapid, feeble pulse. The tongue is uncoated, the mind lucid, and muscular cramps are not uncommon. Constipation is absolute, not even flatus being passed. True fecal vomiting does not *occur* when the obstruction is in the upper third of the ileum, and when high up in the small intestine tympanites does not occur. The quantity of urine passed is much decreased.

In *chronic* intestinal obstruction there is gradual narrowing of the lumen of the intestine, increasing difficulty in securing a bowel movement by the use of laxatives, persistent diarrhea, which is the result of a catarrhal inflammation above the seat of constriction, colicky pains produced by increased peristalsis, pronounced tympanites, and vomiting.

The causes of chronic obstruction include malignant disease at or below the ileocecal valve, cicatricial narrowing affecting the same portion of the bowel, and chronic slowly-progressing invagination. In chronic obstruction there are intervals between the attacks of pain, the exacerbations gradually becoming more frequent and severe. Vomiting, though not of the stercoraceous type, occurs, and the bowel movements are ineffective. In the intervals between these seizures the patient complains of dyspeptic symptoms, suffers abdominal distention, and the attacks of constipation alternate with diarrhea.

These attacks recur with increasing frequency. The patient loses flesh and strength, and may develop marked symptoms of acute obstruction. The subject becomes so emaciated that the distended coils of bowel may be seen beneath the wall of

the abdomen. Borborygmi or bowel noises are common. Death may be caused by progressive exhaustion from a supervening acute obstruction.

### TREATMENT.

If the diagnosis is somewhat doubtful, and the patient appears shocked, a stimulant should be administered, a hot water enema given, and external heat applied. In acute obstruction empty the stomach by lavage, and the bowel by large injections of warm soap water. In acute intussusception, give no food by the mouth. The bowels should be kept immobile with large doses of opium or of opium and belladonna in the form of suppositories. If the intussuscepted intestine cannot be withdrawn because of adhesions, the patient should be etherized, placed in an inverted position, and warm saline solutions of oil injected by means of a fountain syringe, the nozzle being inserted up as far as the sigmoid flexure of the colon.

At this time with the intestines full of liquid, the abdomen should be compressed, care being taken not to exert undue pressure. If this maneuver proves unsuccessful the intestines should be inflated by means of a large indiarubber bag containing air or hydrogen gas, of which 2 or 3 gallons may be cautiously introduced. In cases of intussusception or strangulation of the bowels these efforts must be persisted in for twenty-four hours. If the condition is not relieved the case should pass into the hands of the surgeon. There is a division of opinion in the profession concerning the advisability of introducing a fine trocar and cannula to allow of the escape of gas from the tympanitic abdomen. Many experienced surgeons regard this procedure in minor surgery as a very dangerous expedient. In the statistical table collected by the late Dr. Reginald H. Fitz, 69 per cent. of the cases of intestinal obstruction without laparotomy proved fatal; with operation, 83 per cent. Undoubtedly the high mortality rate under operation can be explained by the fact that in many of these instances surgical interference was deferred too long.

The operation if performed at all should not be postponed longer than twenty-four hours. Pain is to be relieved by morphin, and the incessant vomiting is best met by gastric lavage and the withholding of food for some hours to prevent



retching and aggravation of the symptoms. Gastric lavage may be practised every six hours. Cathartics are absolutely contraindicated in acute intestinal obstruction.

In chronic obstruction treatment is to be conducted on general principles. The bowels should be moved with simple, unirritating laxatives, and due attention paid to the dietary. When there is threatened obstruction, with pain, the treatment just advised for acute obstruction of the bowels should be followed. (See p. 861.) The after-treatment consists in regulation of the bowels, by habit, diet, and aperients, as detailed on the chapter on Constipation (*q. v.*). The employment of massage and electricity, in conjunction with other measures that may suggest themselves to the intelligent practitioner, may prove to be valuable adjuncts of treatment.

### ENTEROPTOSIS; VISCEROPTOSIS; SPLANCHNOPTOSIS.

The term enteroptosis from an etymologic standpoint signifies a falling or ptosis of the intestines, but the condition is of such frequent occurrence coincidently with gastropptosis, nephroptosis, and prolapse of other viscera, including the spleen, liver, and uterus, that the term visceroptosis, splanchnoptosis (Glénard's disease), and enteroptosis are employed synonymously.

In 1885, Glénard published a monograph that attracted wide attention and invited thought from the élite of science; in it he set forth his views upon enteroptosis, and connected therewith certain nervous phenomena which have since borne the name of "Glénard's disease." This masterly article and those that followed by the same writer were widely discussed and commented upon, partly because of the author's earnest enthusiasm and optimism.<sup>50</sup>

"I can affirm," declared Glénard, "that the physician who will follow my directions and strive to verify my statements in such cases will find in his practice the satisfaction which a positive diagnosis gives to both physician and patient, from which alone a proper prognosis can be made, and that satisfaction, the greatest of all, which directs the treatment and avoids for the patient the trial upon him of so many remedies,

while at the same time it secures him relief and prevents the physician himself from falling into therapeutic scepticism." It is an axiomatic saying in medicine that to cure the malady we need seek the cause and attack it. But the theory that accounts for the occasion of visceral ptosis is yet to be pronounced.

Years ago, Stiller declared it to be a congenital anomaly. Schwerdt believed it to be a constitutional malady; an atonic condition of the neuromuscular system. Meinert, of Dresden, ascribed the condition to mechanical causes, and unhesitatingly declared the ill-fitting corset, the constricting waist-band, and other defects of dress, especially among women, to be instrumental in the production of the condition. Charcot regarded the etiologic factor the result of neurasthenia. Sir Frederick Treves offered these theories: Rapid emaciation or severe illness may cause visceroptosis; or it may be engendered by heavy lifting or frequent pregnancies, thereby weakening the abdominal walls and causing a descent of the viscera; or that peritoneal adhesions, by their contracting action might pull down one organ after another. Rosengart regards visceroptosis as a reversion to the fetal or embryonic type; and the elaborate and conscientious study of Arthur Keith regards the condition as "a result of vitiated method of respiration," and he places this complicated entity in the category of respiratory affections.

Each of these etiologic factors is applicable to a number of cases. The theory, as advanced by Keith, supposes that a faulty mechanism of the diaphragm dependent upon many conditions to be the cause of visceral ptosis, for it is a physiological fact that normally the relations of the muscles of inspiration to those of expiration, and the muscular action of the abdominal wall in no way cause or affect, to the slightest degree, a displacement of the viscera. Keith emphasizes the fact that if the integrity of the diaphragm is at all impaired, inspiratory downward displacement is instrumental in bringing about visceral ptosis, especially contraction of the diaphragmatic crura; asserting that although the supports of the diaphragm are three-fold—abdominal, costal and thoracic—that derived from the abdomen, the muscles of expiration, is the most essential to the production of the condition.

Decrease of the sub-diaphragmatic space accounts for displacement of the kidney. This may be occasioned by constriction of the thorax from faulty dress; from deformity and narrowing of the chest by thoracic or spinal disease or permanent contraction of the diaphragm from a relaxed condition of the abdominal wall. The left kidney is not so often displaced as the right, because "the left hypochondrium is provided with a safety valve in the shape of the splenic flexure of the colon"; so that when the sub-diaphragmatic space is decreased, "the colon is extruded and the other organs are undisturbed." Ptosis of the kidney is also largely prevented by the fact that there are intimate attachments between the kidney and spleen, and the spleen and diaphragm. Prolapse of the colon, according to Meinert, is more frequent than gastrop-tosis; the transverse portion, being the most movable, is most often displaced. It may become elongated and tortuous, S- or M-shaped, or found lying immediately above the pubic sym-physis.

A marked degree of visceroptosis may exist without the presence of symptoms. Usually, however, the patient complains of digestive disturbances. He has a sense of fullness after eating, complains of spasmodic pain and flatulence, and vomiting may supervene; as a rule the sufferer is constipated, but diarrhea may alternate with constipation. When gastrop-tosis and nephroptosis are associated, the patient frequently becomes neurasthenic, is irritable, loses flesh and strength, and has no interest in life. Car sickness and train sickness are common symptoms of real moment.

Undoubtedly the largest number of cases is found in women, supporting the theory advanced by Meinert (*q. v.*), and which has been ironically expressed by the gifted writer of "Gates Ajar," in her conscientious efforts to emancipate her sex "from corsets that embrace the waist with a tighter and steadier grip than any lover's arm, and skirts that weight the hips with heavier than maternal burdens."

Persons who are the victims of visceral ptosis do not, as a rule, include those that are fair, fat, and forty, but rather those of a "lean" habit, tall and scrawny, with no compactness of form, the possessors of a sad and doleful expression, indicative of a "neurasthenia basis"; and Keith invites attention to a

peculiar curving of the cervical region, to which he aptly applies the term "ewe's neck."

In a brief exposition of an important condition such as this, one is only able to touch upon its chief diagnostic signs. Suffice it to say that when a patient presents himself, or herself, for an expression of medical opinion, a diagnosis of enteroptosis, or what in reality is the same, visceroptosis, can be quite well established when the following symptom-complex is evidenced. Digestive disturbances (which may not amount to actual pain) both upon arising in the morning and when changing from a recumbent to a standing attitude, a "dragging" under the right costal cartilage, a "giving-way" sensation complained of when the patient turns in bed upon the left side, constipation, lack of ambition, and neurasthenic symptoms, especially in those of a lean habit and of the female sex.

In all of these cases an examination of the abdomen is imperative. The topography of the stomach is to be studied by inflating it with bicarbonate of soda and tartaric acid or by air. The solid organs should be outlined, care being taken to examine the liver in the standing position, since this organ is far more often displaced than is generally supposed.

Glénard, in his elaborate studies, mentions a small band that runs across the abdomen, about 2 inches (5 cm.) above the navel, and is plainly discernible to the palpating fingers; to it he applied the term "*la corde colique transverse*," and regarded it as the "*colon transversum*." But there is good reason to doubt the correctness of this inference, and Boas, Ziemssen and Ewald assert that the cord is the pancreas, rendered palpable by the sinking of the stomach. In palpating the abdomen Glénard lays stress upon the following important test: The patient assumes a standing attitude, and the physician stands behind him. The examiner places both hands flatly over the lower part of the belly and applies even pressure upward and backward. In a large number of these cases much relief is experienced from the distressing dragging sensations felt in the epigastrium and abdomen. X-ray and fluoroscope examination confirm the diagnosis.

## TREATMENT.

If there is a discoverable cause producing or aggravating the displacement, it should, if practicable, be removed. Treatment will be most efficient that will meet all these conditions by the restoration of bodily vigor and by upbuilding of the whole musculature. For these purposes the bowels must be moved regularly, the tonicity of the abdominal walls must be increased by massage, electricity, and hydrotherapy. In cases of neurasthenia appropriate measures must be instituted, including the rest cure. (See vol. i, p. 583.) The food should be nutritious and easily digestible. At times, the employment of gastric lavage acts as an excellent auxiliary to treatment. Drugs are demanded to combat the flatulency and fermentation that are invariably present, and the administration of tonics, as iron, strychnin, arsenic, and quinin are indispensable to the routine of treatment. Belts and supports are useful only when they relieve the intra-abdominal pressure. By their supporting action they encircle the abdomen and exert uniform pressure over the whole abdominal surface. The much vaunted devices advertised to hold a kidney or stomach in place, all are to be avoided as harmful rather than helpful. Autointoxication may be largely prevented by a milk and buttermilk diet.

The indications for treatment as outlined by Glénard are as applicable today as when first pronounced.

(a) The intestines must be elevated and kept in their new position.

(b) The abdominal pressure must be increased.

(c) The bowels must be regulated.

(d) The secretions of the intestinal glands must be increased.

(e) The digestion and nutrition must be regulated and stimulated.

(f) The whole organism must be strengthened.

The anchoring of the kidney, pleating the stomach and gastrocolic omentum, and other measures of raising the ptosed organs may be considered in suitable cases.



## INTESTINAL NEUROSES.

*Secretory Disturbances.* Through a nervous influence the intestinal secretion may be greatly increased in quantity. Clinically, this abnormal secretion may manifest itself as a mucous colic or a pseudomembranous enteritis. The latter subject has been fully discussed under the caption of Mucous Colitis. (See p. 845.)

**Enteralgia.** Enteralgia or neuralgia of the intestines is commonly encountered in hysterical, neurasthenic, or anemic individuals. It occurs as a reflex neurosis in gout and in irritative lesions of the liver and kidneys. It may be a symptom of many affections causing direct irritation of the sensory nerves of the intestines. These include foreign bodies, gallstones, enteroliths, and marked gaseous distention, so that there is increased activity of the motor nerves, or contraction of the muscularis, engendering true intestinal colic.

Enteralgia may occur suddenly, but more often gradually, and is attended with much flatulence. As the attack is fully developed, the pain, which is circumscribed or diffuse, becomes almost unbearable, and may subside quite suddenly. At other times these pains persist for days or weeks, and then gradually vanish. Recurrences are common, but the intervals between the attacks vary greatly in duration.

Hypogastric neuralgia is a term applied to a painful condition encountered most often in the female sex, and in those of a neurotic constitution. It also frequently occurs in cases of tabes and in hemorrhoids. This affection has its seat in the hypogastrium, and provokes distressing pressure-symptoms in the bladder and rectum, the pains radiating to the sacrum, thighs, and perineum.

**Diminished Intestinal Sensibility.** This condition is found quite commonly in affections of the brain and spinal cord associated with paralysis. There is a decrease of peristaltic movements, with more or less anesthesia of the bowels, and a consequent retention of fecal accumulations in the rectum. When the integrity of the motor mechanism of the bowel is not interfered with, and the musculature of the intestines preserves its power of contraction, spontaneous movements of the bowels occur; but when an atonic condition exists, the result

of motor paralysis, the fecal accumulations must be removed by mechanical means.

**Nervous Diarrhea.** Increased irritability of the motor nerves of the bowels results in diarrhea. The condition may be reflex to morbid conditions in the central nervous system or some remote organ of the body. Thus, the affection may be traced to factors such as tabes, certain gastric disturbances, and dentition. It is not infrequently found in persons of an unstable nervous organization, who suffer sudden fright, shock, or surprise. The only characteristic symptoms present are the number of dejections, which may vary from two or three to as many as thirty in the course of the day. The stools follow each other in rapid succession, especially during the morning hours. There are no other constant symptoms.

**Enterospasm.** Spasm of the intestine usually induces spasmodic constipation, and at times total, though temporary, occlusion of the bowels. The etiologic factors concerned in its production are quite analogous to those causing nervous diarrhea, and clinically it offers a study essentially the same as that of enteralgia. Pain or constipation is an inconstant symptom, the stools may or may not be ribbon-shaped, or the patient discharges large round fecal masses, comparable to sheep's dung. The dejecta may be covered with mucus. Proctospasm is usually secondary to fissure or some other rectal affection. In neurasthenic subjects, enterospasm may manifest itself as a neurosis.

**Constipation.** As a functional neurosis, constipation is often found in those suffering from neurasthenia or hysteria, and in victims of various forms of psychoses. Ewald states that these patients are not influenced by the administration of cathartics. Paralysis of the external sphincter is not infrequently associated with diseases of the central nervous system, in which event the dejecta are passed reflexly, due to loss of innervation of the voluntary muscles, or the act may be voluntary but not purposeful, as during mental excitement, sneezing, or coughing, demonstrating merely a condition of bodily weakness.

#### TREATMENT.

In all these cases the best possible hygienic environment and a suitable dietary are to be regarded as more than stereo-

typed text-book suggestions. The treatment of special cases takes into account the nature of the underlying nervous affection and of any pathologic entity provoking the condition, and these need be vigorously combated.

In enteralgia and hypogastric neuralgia the treatment consists in relieving the painful paroxysms by the use of morphin or opium, by the application of heat in some of its many forms; and in the intervals in attending to the alimentary and digestive functions, the administration of tonics, and, if need be, a change of residence. It is asserted by some clinicians that a combination of drugs such as quinin (alterative) and belladonna (antispasmodic) is most effective treatment in the intervals of the attack, and often exerts a curative action. Surely, if such is a fact, the administration of this combination of drugs should merit a trial.

In cases of constipation dependent upon diminished sensibility and atony of the bowel, unless the cause of the complaint can be located and treated, the fecal accumulations must be removed mechanically.

In the treatment of nervous diarrhea, the exhibition of the usual medicaments as used in the ordinary loose dejections of enteritis is contraindicated. The objects in treatment are, first, to relieve pain by the use of the usual anodynes; second, the correction of the causes on which the attack depends. The large serous discharges that are likely to occur as the result of anxiety or other emotions are best met by prescriptions containing camphor, chloroform, and the volatile oils. All active exercise must be interdicted. Heat may be applied to the abdomen, and the food should be restricted to broths or milk foods. When the number of stools passed is large the patient should take to bed. The treatment of enterospasm is identical with that of enteralgia (*q. v.*).

## CARCINOMA OF THE INTESTINE.

This is a very common cause of intestinal obstruction. It is more common in men and in persons over the age of fifty. The etiologic factor in very many instances is dependent upon the local irritation produced by the friction of the fecal mass against the intestinal walls, and finds corroboration in the

study of statistics of diseases of the large intestine, where 80 per cent. of cases of cancer have been found in the rectum, the remaining 20 per cent. distributed in the cecum, and in the sigmoid, splenic, and hepatic flexures of the colon.

Cancer of the bowel may exist for some time without suggestive symptoms directly referable to the intestine until ulceration, stricture, or tumor is manifest. Previous to this, and extending possibly over a long period of time, the patients bowels have been "irregular." Later there is a sense of discomfort, but as the condition progresses this discomfort asserts itself in the form of colicky pains, which gradually increase in intensity. When the rectum is invaded, the characteristic tenesmus is a cardinal symptom.

The patient's loss of flesh and strength are out of proportion to the suffering experienced, and although the victim of the malady may have a normal appetite and good digestive powers, the resulting cachexia is an unfailing sign of a malignant onslaught upon his vital powers. When the cancerous deposit is low down in the large intestine the fecal discharges conform to the narrowed caliber of the tube, and are ribbon-like or furrowed, and blood and mucus, as well as portions of tissue and pus, may be made out both by macroscopic and microscopic investigation. With the onset of these phenomena, the raw and bleeding surface of the bowel occasions continuous diarrhea and intense pains, the patient suffers marked exhaustion, anorexia asserts itself, the digestive powers are impaired, and there is blanching of the skin, and the facial expression is one of marked anxiety.

Late in the affection, the presence of a visible and palpable tumor is part of the course of this fatal invasion, the patient usually not surviving the attack longer than six months to a year. A speedy fatal issue follows perforation, which at times takes place. Death, as a rule, results from emaciation and exhaustion, with edema of the lungs as a terminal symptom.

#### TREATMENT.

From a strictly medical standpoint, treatment of cancer of the intestines is absolutely palliative. The diet should be of the most nourishing character and easily digestible, but when symptoms of obstruction assert themselves the ingestion of

food by the mouth is interdicted. In cases of duodenal or jejunal carcinomata, nutrition should be effected by means of nutritive enemata. For the violent pains subcutaneous injections of morphin and also opium by suppositories should be given. *Cannabis indica*, with or without opium, is often productive of good results. When symptoms of marked depression are evidenced, the administration of stimulants is demanded. The bowels must be kept in a soluble condition by laxatives and enemata, and lavage of the stomach offers great relief to the patient, in ridding the system of fermenting food, which not infrequently is retained in the stomach or regurgitated into that viscus. If there are evidences that a stricture has reached an advanced stage, the irritating action of laxatives of all kinds had better be dispensed with, and enemata resorted to, as the action of aperients may cause perforation and rupture. Further treatment in these cases belongs to the domain of the abdominal surgeon.

### TUBERCULOSIS OF THE INTESTINE.

Next to the lungs, the intestines are the most frequent seat of tuberculosis. Chronic diarrhea is a cardinal symptom of the disease, often alternating with temporary periods of normal movements, or even with constipation. The dejecta vary in color according to the drugs that have been given, and the ingesta consumed, and are extremely feculent and slimy, and may contain blood. The movements of the bowels often are excited by food or drink, the discharges being preceded by colicky pains that are relieved by evacuation. Intestinal tuberculosis as the primary lesion of this infection is far more common in childhood than in adults. When thus affected the child appears pale and wan and has a protruding abdomen as the result of gaseous distention. The little one's skin is harsh and dry, and hangs from its body in wrinkles and folds, giving it the appearance of a withered old crone. The distended belly usually is not tender, the tension being relieved by the escape of gas, at which time the mesenteric glands may become visible. The lymphatic glands in the groin, axillæ, and neck may become enlarged.

The temperature changes conform to the usual character



of fever in tuberculosis, and during the exacerbations profuse sweating, especially of the head and back, is commonly observed.

Secondary tuberculosis of the intestine is frequently combined with pulmonary tuberculosis in both young and old. The appearance of the tubercle bacillus in the dejecta is not of diagnostic import, because of the frequency with which the sputa are swallowed. If diarrhea is present it stubbornly resists treatment, and it should be remembered that this troublesome symptom may be produced by a catarrhal colitis or amyloid change, both of which processes may occur in the course of pulmonary phthisis. Abdominal pain is usually inconsiderable, although attacks of colic occur. The abdomen is retracted, and tender spots may be elicited upon pressure, showing the extension of the ulcerative process toward the peritoneal surface of the bowel. The chief location of the infective lesion may be for a period of time at the cecum or in the appendix, when both local and general symptoms of appendicitis are a conspicuous element of the affection.

#### TREATMENT.

The treatment of intestinal tuberculosis must have for its basis the general hygienic management and the treatment as applied to cases of chronic tuberculosis. A most important factor in the treatment of the diarrhea is a properly restricted dietary. Among the more useful medical measures, used singly or in combination, are bismuth in large doses, acetate of lead, opium, thymol, salol, silver nitrate, creasote, and chalk mixtures containing tannic acid. Externally, turpentine stupes, mild counterirritation, and the application of spice plasters, often afford relief.

#### SYPHILIS OF THE INTESTINE.

The intestines may become the seat of syphilitic lesions similar to those affecting other mucous surfaces. Gummata, diffuse infiltrations of the intestinal canal, and perforating ulcers probably due to gummata have been diagnosed and reported by many clinicians and syphilographers.

The rectum may become the seat of a series of important

changes due to this disease. Women are more prone to intestinal syphilis than men, in the proportion of 8 to 1. This is in part due to the anatomic differences of the sexes, to the occurrence of the menstrual molimen in women, to previous pregnancies, and to unnatural and excessive coitus.

Care must be taken to distinguish between the induration of the submucous tissues about the rectum with purulent sanguinolent discharge and constipation due to chancroids and syphilitic stricture of this part of the bowel. The most important syphilitic affections of the rectum are those characterized by ulceration or gummatous changes; the former may extend from the perineal region to an inch (2.5 cm.) or more within the sphincter. The ano-rectal syphiloma is a non-ulcerative gummatous infiltration of the anus and the walls of the rectum, often resulting in stricture, the result of transformation into fibrous tissue.

#### TREATMENT.

To attempt to outline the treatment of this special form of syphilis would be a work of supererogation, for the management of lues has been described elsewhere in minute detail. (See vol. i, p. 78, *et seq.*) In addition, the usual hygienic measures are to be followed, as laid down in the treatment of all constitutional ailments. The diet should be nutritious, attention paid to movements of the bowels, and tonic treatment administered when occasion demands.

Drug medication includes inunctions with mercury, the administration of mercury and chalk, especially in children; the employment of mixed treatment, the latter often combined with one of the forms of iron, a routine followed by many excellent practitioners, who assert that such a ferruginous addition prevents the development of stomatitis and salivation.

In place of the administration of mixed treatment, many physicians believe in the efficiency of the iodids or preparations of mercury when given alone; although so great an authority as the late Prof. J. Wm. White and a large following of expert syphilographers emphatically deny the possibility of curing tertiary syphilis by the administration of the iodids alone.

Much attention has been given in recent years to the intra-

venous injection of salvarsan or arsenobenzol, and also of sodium cacodylate, by the intermuscular route. Advantage is taken of the injection of mercury salicylate, to be dissolved in warm water and given intramuscularly in doses of 0.065 gram (1 gr.); the use of succinate of mercury and gray oil may supplement these procedures. The regulation of the dose and the frequency of application depend upon the symptoms and the physiologic effects that are manifested. From time to time the Wassermann reaction is to be taken as a corroborative measure of the efficiency of the medication instituted.

## DISEASES OF THE LIVER.

### GENERAL CONSIDERATIONS.

The liver may be rightly considered the filtration plant of the abdomen. Its circulation is such that the hepatic function is closely related to all of the abdominal viscera. Changes in the organs may be accompanied by changes in the liver or *vice versa*. There is also a direct relation between its sympathetic supply and the general nervous system, which when inco-ordinate may act reflexly upon the normal functions of the liver. Undue nervous excitement, fatigue, certain industrial employments, personal habits, intoxications, infections, and in fact any physical, chemical, mechanical, or physiologic disturbance of the body may affect the hepatic functions. Disturbances of the ear and of the eye may also affect this organ indirectly.

A brief discussion of the anatomy and physiology of the liver may be considered of advantage in making clear its pathologic lesions. This organ is made up of four systems of vessels—arteries, portal and hepatic veins, and bile ducts. Each or all of these may be subject to irritation or to obstruction as the result of interference with its normal physiologic function or to disturbances in adjacent organs. Cardiac and renal disease have a direct influence upon the liver. Being an important organ of protein metabolism, it is possible to determine the extent of the disease by urea production. It is the cradle and the grave of the red corpuscle, and is directly concerned with sugar digestion, and in the latter capacity is

the storehouse for carbohydrates. It produces bile, an essential product of intestinal indigestion.

Because of its special anatomic relation between the return circulation from the liver and the inferior vena cava, the liver is subject to the influence of interference with normal circulation caused by cardiac insufficiency. Various hepatic derangements may, therefore, be expected in cardiac lesions.

The liver receives the return blood from the stomach, spleen and intestines, and is therefore subject to irritation caused by various products resulting from deranged function of these organs. Certain chemical substances have a special predilection for the liver when taken internally. Disintegration, degeneration, and irritation may result from the influence of such preparations as phosphorus, turpentine, chloroform, ether, mercury, and explosive chemicals.

Again, the liver because of its intimate relation with the intestinal tract, may harbor the infant or adult forms of various parasites, such as the dog tapeworm, flukes, ankylostoma, oxyuris, and others.

Constant irritation leads to the formation of excessive connective tissue at the expense of the venous supply, causing a damming back of the blood into the intestinal viscera, producing a chain of symptoms described later under *cirrhosis of the liver*.

It is thus seen that diseases in other organs may produce changes in the liver, or that diseases originating in the liver itself may produce symptoms in other organs. In other words, when the hepatic function is deranged, the symptoms are not only referable to this organ but to the whole body.

## BILIOUSNESS.

This common phraseology is applied to the symptom-complex characterized by nausea, usually with vomiting, coated tongue, anorexia, headache, constipation and yellowish or icteroid complexion of the skin, and a feeling of weakness or indisposition for mental work. It occurs after indiscretion in diet, after the ingestion of certain foods known to disagree with the individual, excessive indulgence in alcoholic bever-

ages, work in a vitiated atmosphere, fatigue, undue excitement, and inattention to personal hygiene.

### TREATMENT.

The first indication in the way of treatment is to obtain a free and satisfactory movement of the bowels. A course of calomel taken in quantities of  $\frac{1}{6}$  or  $\frac{1}{4}$  grain (0.01080 or 0.01620 Gm.), repeated every half-hour until 1 grain (0.065 Gm.) has been taken, followed one hour later by a saline purge of Epsom salts, citrate of magnesia, or Glauber's salts, may clear up the condition without further medication. In adult males of middle life satisfactory results may be obtained by the administration of 1 or 2 compound cathartic pills at bedtime. In spite of their griping action, they seem to move the most stubborn forms of constipation and relieve the patient of all toxic products. Nausea often may be relieved by the ingestion of plain seltzer water, taken ice cold, or with the addition of sodium bicarbonate or sodium phosphate.

The indiscriminate use of coal tar products and of patent preparations for the relief of headache during attacks of biliousness is severely condemned in that they do not remove the cause of the predominant symptoms. There are many cases on record of poisoning resulting from the careless use of pills for liver trouble.

The prevention of repeated attacks calls for especial attention to personal habits. The use of a drastic cathartic at least once a week by persons who are constantly constipated is of great value. Persons who lead a sedentary life, who are generally employed indoors, and who obtain very little exercise, should make it a point to adopt some form of physical calisthenics in the early morning hours before breakfast, or at least walk to or from work if possible. In various industrial pursuits it is necessary for the worker to assume attitudes and positions which cramp the abdominal viscera, thus hindering normal peristalsis. In various establishments employing girls and young women, accommodations for their personal needs are inadequate or undesirable, causing them to become indifferent to their natural inclinations until they arrive home in the late evening hours. It is the experience of many physicians to receive complaints from patients to the effect that



they have not moved their bowels for three or four days, and sometimes a week, resulting in chronic constipation and frequent attacks of biliousness. Persons working under such conditions should make an effort to have a daily movement of the bowels every morning or every evening. If the bowels are sluggish, it is advisable to take 2 pills consisting of aloin, belladonna, strychnin, and cascara, U. S. P., at bedtime, and repeat it until the bowels become regular. Of late, mineral oil has become quite popular in facilitating regular passage of the bowels, and may be administered night and morning, a half-ounce (15 mils) each time. Of greatest importance, however, in regulating the bowels is the diet. Liquid foods should be taken in plenty. Vegetables and stewed fruits assist in causing semi-solid movements. Persons subject to biliousness should avoid shell-fish, fatty foods, rich pastries, and smoked meats. Persons of neurotic tendency or subject to so-called nervous biliousness should be placed under the influence of bromids.

### JAUNDICE.

Jaundice is a symptom-complex characterized by the staining of the skin, conjunctivæ, and the secretions of the body (saliva, urine, and sweat) with bile pigments. It is caused by obstruction of the normal flow of bile in the liver, in the common bile duct or in the duodenum. Inflammation of the common bile duct and duodenum is probably the most frequent cause. Among the obstructive agencies are gall-stones, parasites, tumors, displacements of viscera, enlarged glands, floating kidney, and pregnant uterus. The stagnant bile is absorbed by the general blood-stream, resulting in staining of the tissues of the body, both internally and externally, while the chemical properties of the bile have a depressing influence upon the nervous system.

### CATARRHAL JAUNDICE.

This condition arises from a catarrhal condition of the bile-ducts and duodenum in the immediate vicinity of the ampulla of Vater, producing general discoloration of the surface of the body, and attended with various nervous symptoms.

The liver is slightly enlarged, lighter in color than normal, and bile-stained. On section, the bile-ducts are patulous and distended with bile. The surface of the cut section is stained, the common duct is swollen, its mucous membrane thickened, and its lumen filled with viscid bile, which can be expressed by pressure. Section of the duodenum will also reveal a hyperemic condition of the mucosa. Microscopically, the liver cells are dotted with bile pigment, imparting a dark greenish color. In marked cases the pigment collects in irregular masses in the bile capillaries. The pigmentation is most marked in the central zone of the lobules.

Marked constipation is a frequent predisposing cause. Overeating, improperly cooked and poorly masticated food, excessive use of alcohol, coffee and tea, mental and physical fatigue, exposure to wet and cold, certain infectious diseases (pneumonia, typhoid fever, malaria), and cardiorenal disease with failure of compensation, are among the many exciting factors. The inflammation begins in the duodenum and travels upward through the common duct.

Jaundice is the most frequent symptom, affecting the entire skin surface and conjunctivæ, the latter presenting a lemon-yellow color. The urine and sweat are often discolored, perhaps so decidedly as to stain the bed-linen. The urine varies from a greenish yellow to dark brown. When thoroughly shaken in a bottle, a yellow foam appears on the surface, while bile often may be detected in the urine before it affects the skin and conjunctivæ. In cases of moderate duration, albumin and hyaline casts, the latter often bile-stained, may be found. The stools lack their normal color, being pale drab or slate. The bowels are costive, but later may be very loose. Occasionally the tears and saliva are stained. The temperature may be normal or slightly elevated (100° F. to 101° F.—37.7° C. to 38.3° C.), while the pulse is slow but full (as low as 20 to 30). There is tenderness over the hepatic area, and paroxysms of pain, which may be severe or slight, and accompanied by nausea, sometimes vomiting, headache, and prostration. Itching is a common symptom. Urticarial eruptions may appear upon the body. Free sweating is frequently complained of; it may be either general or localized to the abdomen and the palms of the hands. In various cases

of jaundice, hemorrhage may occur beneath the skin and mucous membranes. The toxic effect of bile absorption causes a feeling of mental and physical depression, headache, and fatigue. When existing with carcinoma, acute yellow atrophy of the liver, and fatty degeneration, the nervous symptoms are intensified, approaching delirium, and often-times causing convulsions or coma, which may terminate fatally. The vision may become affected, the patient complaining of colored objects before the sight.

On examination the liver is found to be enlarged, the degree of the hepatomegaly depending upon the duration and severity of the bile obstruction. The border of the liver may reach several inches below the costal edge. Palpation or percussion over the liver and gall-bladder area elicit tenderness and cause pain, commonly referred to the back. The disease may last for from two to eight weeks. Cases prolonged beyond this period may be rightfully considered as other than simple catarrh. The prognosis is favorable. When, however, there is a continued rise of temperature, or when there are subcutaneous and submucous hemorrhages, the prognosis is less favorable.

#### TREATMENT.

If the pain is severe, it is relieved by hypodermic injection of morphin,  $\frac{1}{4}$  grain (0.01620 Gm.), repeated in a half-hour if the suffering demands it. The patient should be kept in bed and hot compresses placed over the hepatic area for twenty minutes in every hour. The bowels may be moved by the administration of compound jalap powder, in a dose of from 30 grains to 1 dram (1.95 to 3.9 Gm.) at bedtime. In the morning effervescent solution of phosphate of soda, 1 dram (3.9 Gm.) to half a glass of hot water given a half-hour before breakfast, is quite acceptable. If the bowel movements are not satisfactory, a dose of citrate of magnesia should be given. If the attack is a mild one and there is no fever and the pain has subsided, the patient may be allowed out of bed and the treatment continued. Chronic constipation calls for repeated enemata, using simple salt solution, soap suds, or emulsion of asafetida. A tablespoonful of milk of magnesia (15 mils) should be given at bedtime to soften the stool.

The diet plays a very important part in the treatment of

catarrhal jaundice. Milk alternated by buttermilk or skim milk should constitute the main diet for the first two days. Ice is quite acceptable in allaying nausea. Although acids in general are contraindicated, the juice of sweet oranges with shaved ice makes a palatable drink, allays the thirst, clears the stomach of mucus and bile, and sweetens the taste. A draught of plain ice-cold seltzer water is also acceptable in that it assists in dissolving the mucus in the stomach, encourages peristalsis and dilutes the toxic products. All fats and rich pastries are to be prohibited. As the jaundice disappears there may be added to the diet strained beef broth, beef tea, milk toast, gelatin, custard, and tapioca pudding; later, chicken, squabs, and boiled beef are permitted. Stewed prunes may be added, as well as baked apples and cream. In spite of the fact that the liver is specially concerned in sugar metabolism, a small amount of carbohydrates in the diet is permissible. A full diet is given only after the jaundice has completely disappeared.

Among the drugs which are valuable are sodium salicylate and sodium succinate, each 3 grains (0.195 Gm.) in capsule three times a day; sodium bicarbonate, 10 grains (0.65 Gm.), potassium citrate, 10 grains (0.65 Gm.), or dilute hydrochlorate acid, 10 minims (0.65 mls), given in tincture of gentian, cardamom or essence of pepsin, as a routine measure. Effervescent phosphate of soda may be taken daily, 1 dram (3.75 mls) to a half-glass of hot water a half-hour before breakfast. A full tub bath should be taken daily in order to aid in eliminating the toxic products through the skin and to stimulate the circulation with a view of causing a return of normal function of the liver. Itching may be controlled by the application of a 2 per cent. solution of menthol in alcohol. Bromids are advocated in abating the nervous symptoms. Should the jaundice recur or be prolonged, colonic irrigation of the bowel is advocated, using from 2 to 3 liters (2 to 3 qts.) of salt solution at each *séance*. This dilutes the toxic products, stimulates the portal circulation, and favors normal peristalsis. If after the acute symptoms have subsided the jaundice still lingers on, a course of calomel given for two or three days, in doses of  $\frac{1}{10}$  grain (0.00648 Gm.), followed by Epsom salts or citrate of magnesia, may assist in favoring the abatement of

symptoms. Jaundice caused by pressure of neoplasms, gall-stones, or similar causes calls for operative interference and drainage of the gall-bladder.

### ACUTE INFECTIOUS JAUNDICE.

Surgeon-General Blue, U. S. Public Health Service, has granted the authors permission to reproduce under *Diseases of the Liver* the subject matter of acute infectious jaundice, as published by the U. S. Public Health Service in its Report of May 10, 1918, by M. H. Neill, Passed Assistant Surgeon. Since the material furnished in this article contains the latest information on the subject of acute infectious jaundice, common among the troops in France, and occurring in scattered areas throughout the United States, it is deemed best to repeat this article as originally published:—

“Acute infectious jaundice is an acute infectious disease characterized by malaise, prostration and gastro-intestinal symptoms at onset, by fever of varying degree and by jaundice of varying intensity and duration. In severe cases bleeding from mucous surfaces and albuminuria are common. In moderately severe cases the rather high fever, marked prostration and absence of local signs tend to exclude local disease of the biliary tract, and present the clinical picture of an acute infection. Light cases of this affection, however, seem to be clinically indistinguishable from ordinary catarrhal jaundice, and therefore are seldom diagnosed correctly in the absence of an outbreak of the disease, which naturally directs attention to the probably infectious character of the malady.

*Prevalence of the Disease Among Troops in Europe.* It is well known that outbreaks of jaundice have occurred at different times among the French, British, Italian, German and Russian troops. The geographical range of prevalence has been from Belgium to Gallipoli. In most of the outbreaks the mortality has been low, but in some of the commands the attack rate has been high.

At this time the weight of evidence indicates that the disease in the great majority of instances, if not in all, has been due to the *Spirochæta icterohæmorrhagiæ* of Inada (1916) and his co-workers. This organism was first demon-



strated as the cause of a severe form of the disease prevalent in Japan.

*Prevalence of the Spirochæta Icterohæmorrhagiæ Among Wild Rats in the United States.* Noguchi (1917) found that rats captured about New York City were infested with a spirochæta identical in appearance with that causing acute spirochætal jaundice in man. This worker, by means of cross immunity tests, presented further evidence that the parasites causing the human disease in Europe and Japan and those found in New York rats were the same. Jobling (1917) found that of more than a hundred rats captured in Nashville, Tenn., at least 10 per cent. carried similar spirochætes in their kidneys. The writer has found a similar prevalence of the *Spirochæta icterohæmorrhagiæ* in wild rats captured in Washington, D. C. As far as can be made out the organisms found by different workers in the United States correspond very closely in appearance and pathogenicity for guinea pigs with those infecting human beings in the trenches in Europe and in the mines of Japan. The evidence then seems to indicate pretty conclusively that the *Spirochæta icterohæmorrhagiæ*, the cause of acute spirochætal jaundice in man, is rather widely disseminated among wild rats living under such different conditions of environment as obtain in New York, Washington, and Nashville. Reports of the degree of prevalence of these parasites in the rats throughout the country are awaited with considerable interest.

*Reported Occurrence of Epidemic Jaundice in the United States.* A search of the literature reveals a number of outbreaks of jaundice occurring from time to time in this country. Even though few and far between, on account of the prevalence of the causative agent in wild rats, and the fact that outbreaks of jaundice tend to occur among troops, these reports deserve at least a passing consideration.

An account of an outbreak of jaundice among troops in the War of 1812 has come down to us. Acute infectious jaundice has been stated to have been highly prevalent during the Civil War, and various numerical estimates of its prevalence appear in the literature. The following quotation is taken from the Medical and Surgical History of the War of the Rebellion prepared under direction of the Surgeon-General, United States Army: 'Jaundice occurred frequently in the progress of the malarial or

other fevers as the result of morbid changes affecting the liver or blood. The yellow coloration in these cases was mostly an incident or symptom of the well-defined primary disease. There were, however, a large number of hepatic or hæmatic disorders in which the alteration of color represented so prominent a symptom that the disease was recorded under the heading of jaundice. Not less than 71,691 cases of this kind were reported among white troops (Union Army). Generally the cases were sporadic, but sometimes a series occurred in a command constituting a local epidemic.'

While in the report just quoted the association of jaundice with disease of the liver and malaria is recognized, the records contain several accounts of clear-cut outbreaks of jaundice and fever corresponding pretty closely to the descriptions of the trench jaundice observed in the present war. It seems, then, that, while outbreaks of acute infectious jaundice, very likely due to spirochætes, occurred during the Civil War, a numerical estimate of the prevalence of the disease should not be attempted.

A search of the literature reveals a number of reports of outbreaks of jaundice among the civil population in the United States. In the majority of these reports no special prevalence of other febrile diseases is mentioned in connection with the cases reported. In many of the cases the observers were impressed with the fact that they were dealing with a condition they had never seen before, basing their diagnosis on the description of the disease as it occurs in Europe.

In all the American reports most of the patients experienced nausea or vomiting, some abdominal distress or pain, headache, and fever of varying degree, followed in a few days by jaundice of varying intensity. It fact from many of the descriptions there is little to suggest a specific infectious disease, aside from the fact that a number of cases as described above would appear at about the same time in a community which was both previously and subsequently free from the disease. Several reports indicate a high mortality among pregnant women. In some outbreaks children seem to have been chiefly attacked, in others adults, and in still others adults and children were equally affected. In some outbreaks males were principally affected, while in others both sexes were affected about equally. In several outbreaks the symptomatology in the fatal cases was strik-

ingly similar, suggestive of rapid necrosis of the liver cells as occurs in acute yellow atrophy of the liver. There is no information as to the occupation of those ill with the disease, nor is it possible to gain any comprehensive idea of the sanitary situation as regards water and food supply, sewage disposal, and the like, under which the disease has occurred in the United States. No adequate study of the pathology of the disease in man in this country has come to the writer's attention. Barker and Sladen found that the blood serum of their cases agglutinated a strain of *B. paratyphosus*.

There is some evidence to suggest that direct contact may have occasionally played a part in transmitting the disease, as in Hanover, N. H. (Gile, 1908), where a number of college students were engaged in surveying roads in the vicinity, camping out at night. From time to time one would become ill with jaundice and fever and return to college. Then cases began to appear in the college itself where the disease had formerly been unknown. Again, at Ann Arbor, Mich (Cummings, 1915), 12 of the 19 cases had been in contact with a sick college mate previous to contracting the disease. In this outbreak the food supply was apparently not a factor, as nearly all those attacked ate at separate boarding houses.

From the table (p. 885) it is evident that, while in certain of the outbreaks a fairly large proportion of the community has been attacked, the case-fatality rate has always been low. As regards seasonal prevalence, it would seem that the fewest cases occurred in the warmer months of the year. Detailed analysis of the reports shows very few cases occurring in the summer as compared with the fall and winter. This is in accordance with a part of the observation of Japanese and European workers, that the disease does not occur in the hottest or coldest weather.

Before closing the discussion as to the prevalence of epidemics of jaundice in the United States it seems fair, in the absence of more definite knowledge, to ask whether such a disease as a separate entity has existed or whether all the outbreaks were not manifestations of some other disease such as typhoid fever or malaria. With regard to malaria it may be said that, while estivo-autumnal malaria undoubtedly caused outbreaks of jaundice in the Civil War, it is contrary to our present knowledge of the distribution of this disease to ascribe such outbreaks

as occurred in Maine, New Hampshire, Minnesota, and Wisconsin to this cause. To infer that these cases were due to some vagary of *B. typhosus* would contradict a great mass of clinical experience, which shows that jaundice is a very rare symptom of typhoid fever. The same may be said with regard to paratyphoid infection. In general the seasonal prevalence of acute infectious jaundice seems to be at its lowest just when so-called filth-borne diseases are most prevalent.

Whatever may be the weight attached to such facts as have been stated above they indicate that epidemics of jaundice closely simulating those now known to be caused by the *Spirochæta icterohæmorrhagiæ* have occasionally appeared in this country, and that they were possibly due to this parasite. This latter statement is strengthened by the finding by A. M. Stimson (personal communication) of spirochætes in sections of the kidney of a man who died in New Orleans of a disease characterized by jaundice and fever. These sections prepared by Levaditi's method show spirochætes morphologically similar to the causative agent of acute spirochætal jaundice.

*The Problem of the Rat as a Carrier of the Spirochæta*

Place.	Approximate population.	Year.	Months.	Cases.	Deaths.
Rocky Mount, N. C. ....	?	1849-50	Nov., Dec., Jan. ....	About 40....	Not stated
Halifax Court House, Va. .	?	1857-58	Fall and winter .....	Not stated	Apparently none.
Savannah, Ga. ....	30,000	1880	Jan., Feb. ....	80 .....	None
Birmingham, Ala. ....	3,000	1881-82	Sept.-Jan. ....	"Many" ...	"Few"
Plainfield, Mich. ....	100	1886-87	Dec., Jan. ....	22 .....	None
Geneva, N. Y. ....	7,000	1888	Spring .....	200 .....	None mentioned.
Troy, Me. ....	?	1887	.... do ....	Not stated	do
Sparta, Wis. ....	3,000	1898	Aug., Sept., Oct. ....	"Few" .....	"Few"
Calumet, Mich., and vicinity .....	30,000	1897-98	June-Jan. ....	675 .....	None
Hanover, N. H. ....	4,000	1899	Not stated ..	About 25 ..	do
Stirling, Kans. ....	2,200	1905	Sept., Oct., Nov. ....	30 .....	2
Montevallo, Ala. ....	.....	1906	Nov., Dec. ....	Not stated	None
Talladega, Ala. ....	5,500	1907	Summer and fall .....	About 200 ..	2
Baltimore, Md. ....	558,500	1908	Nov., Dec. ....	6 cases, of 700 inmates of jail ....	None
Andover, Me. ....	750	1908-09	Oct.-Feb. ....	135 .....	do
Austin, Minn. ....	6,960	1910	Sept.-Dec. ....	About 200 ..	do
New York City .....	4,800,000	1912-13	Oct.-Jan. ....	25 studied at author's clinic .....	1
Hetland, S. Dak. ....	223	1913	June-Sept. ....	Not stated	Not stated
Ann Arbor, Mich. ....	15,000	1915	Spring .....	25 .....	None



*Ictero-hæmorrhagic.* The following quotation from Noguchi is well adapted as a starting point in this discussion: 'The finding of the causative organism of infectious jaundice among wild rats in America and the identification of this strain with those found in Asia and Europe seem to be particularly important in revealing a latent danger to which we have been constantly exposed, but from which we escape as long as sanitary conditions are not disturbed by untoward events.'

Long before the present war acute infectious jaundice was recognized to occur especially among troops, among sewer workers, agricultural laborers working in wet soil, and in mine workers. People who handle food as butlers and cooks, in Japan at least, are also said to be attacked with especial frequency. *With the universal adoption of trench warfare in the present conflict acute infectious jaundice took a more or less prominent place in the category of trench diseases. Stokes (1917) observed a definite increase in the number of cases among troops during wet spells of weather, followed by a diminution in cases when the weather became dry.* It was also noted that a regiment which had a number of cases in the line—i.e., wet trenches—was not infected while in rest billets, but again produced cases when it returned to the trenches. In Japan, Inada (1916) and his co-workers found that cases of acute infectious jaundice occurred in the wet shafts of the mine, but not in the dry shafts nor on the surface. Some evidence has been presented to show that the hot and cold months of the year are unfavorable to the spread of the disease. Several laboratory workers have been directly infected by the blood of guinea pigs suffering from the disease, at least once with fatal outcome.

The credit of first finding the *Spirochæta ictero-hæmorrhagicæ* in rodents belongs to the Japanese investigators, who first demonstrated these parasites in the kidneys of field mice. Further investigations in the coal-mining regions of Japan showed that 40 per cent. of the wild rats harbored organisms resembling these parasites. Many cases of infectious jaundice in human beings, due to spirochætes, occur in this region. With regard to the spirochætes found in the rats, it was observed that they live in the kidney without injury to the animal, and are excreted in the urine. By means of tests with immune sera, evidence was obtained which indicated that the spirochætes which came from the



rats were quite similar to, if not identical with, those derived from human sources. The various strains of these spirochætes all produced the same striking pathological picture in experimentally infected guinea pigs. Guinea pigs were infected by allowing rats to bite them, and it was demonstrated also that the organisms would pass through the unbroken skin of these animals.

English and French workers soon demonstrated the presence of the *Spirochæta icterohæmorrhagiæ* in rats taken from the trenches in which the disease had appeared among troops. Thus Stokes (1917) found 6 out of 15 rats to be infected. On the other hand, Courmont and Durand (1917) examined 50 rats taken in a region where acute infectious jaundice was unknown. The rats appeared perfectly healthy, but four of them were proved by guinea-pig inoculation to harbor the *Spirochæta icterohæmorrhagiæ*. These figures approximate the rate of incidence later obtained for wild rats in the United States. Rat infestation has been demonstrated in other portions of France.

With regard to the relation between rat infestation and human infection with *Spirochæta icterohæmorrhagiæ*, two possibilities present themselves. First, it is possible that no transfer from rats to man takes place, or only exceptionally, as in case of a bite. It may be that some cause is at work in the trenches and mines which tends to infect man and rats with the *Spirochæta icterohæmorrhagiæ* entirely independently of each other. On the other hand, it seems more probable that the spirochætes may be interchanged indiscriminately among men and rats living in such environments as obtain in the trenches, by means of their urine. There is evidence to show that infection can take place either through the skin or by the mouth. There is no adequate evidence that any insect plays a part in the transmission of the disease in nature, although the experimental evidence in this regard is by no means complete. The epidemiology of the disease seems to point rather definitely to a moist soil, at an equable temperature, as a means of keeping alive the virus.

While the problem of the rat in relation to acute infectious jaundice has not been completely worked out, the following statement of the mode of transfer forms a reasonable hypothesis. About 10 per cent. of all wild rats wherever located probably carry the *Spirochæta icterohæmorrhagiæ* in their kidneys and excrete them in their urine. If this organism finds a favorable en-

vironment in the soil, a sufficient number may live long enough to infect a human being who gets them in the mouth or on the skin. Under these conditions a larger number of rats also take up the spirochætes.

Much more work needs to be done to place the whole matter on a sound scientific basis, and to do this it is essential to (1) recognize cases of the disease in man, (2) determine the general prevalence of the *Spirochæta icterohæmorrhagiæ* in wild rats, living in various environments.

*Detection of the Spirochæta Icterohæmorrhagiæ by Laboratory Methods:*

(a) *In man.*

The following methods have been successfully employed in detecting infection with the *Spirochæta icterohæmorrhagiæ* in human beings:

1. Examination of blood films. These have been stained for spirochætes by one of the Romanowski stains or one of the silver impregnation methods.

2. Examination of the blood by dark field illumination.

3. Injection of the blood into the peritoneal cavity of a guinea pig.

In these three methods, the earlier in the course of the disease the blood is obtained the better the chances of success. In the first two methods search must be made with the microscope for the spirochætes; and as they are not very numerous in the blood of human cases, and somewhat difficult to stain, these methods are not highly satisfactory. On the other hand, in early cases guinea-pig inoculation with blood is a valuable procedure, and should always be done, unless the patient is first seen late in the disease. If the *Spirochæta icterohæmorrhagiæ* are present in the inoculated blood the guinea pig will usually sicken and die in about ten days. *Post-mortem* examination will show a well marked combination of jaundice and hemorrhage such as, so far as known, is not produced by any other infection.

In a light-skinned guinea pig a distinct yellowish tinge, especially noticeable in the ears and about the genitals, is usually observed. On dividing the skin of the abdomen in a case of this disease, the operator is at once struck with the widespread hemorrhages which lie beneath the skin and between the connective tissue planes. They range from minute petechiæ up to

massive effusions of blood perhaps a centimeter in diameter. The hemorrhages are especially well marked about the axillary and inguinal lymph-nodes, and as the skin is reflected hemorrhagic areas will be seen between the fascia covering the skeletal muscles. The skin is usually quite yellow and the abdominal muscles frequently show a yellowish tinge. On opening the body cavity the liver appears distinctly enlarged and of a brownish-yellow color. The spleen is not enlarged. The intestines are stained yellow, and hemorrhages into the intestinal walls are of frequent occurrence. Post-peritoneal hemorrhages are frequent and abundant, especially about the kidney and adrenal. This organ is frequently the seat of marked effusions of blood. In the thorax the lungs especially attract attention, being the seat of the most characteristic gross change observed in the guinea pig. These consist of numerous sharply defined hemorrhagic foci. The description, by the Japanese, of the lungs as resembling the mottled wings of a butterfly is a very apt one. Histologically the liver and kidneys show the most characteristic changes. The liver shows an exudation of polymorphonuclear leucocytes about the bile-ducts, and widespread degenerative changes of the parenchyma. Many of the cells contain an abnormal amount of pigment, while others show pronounced vacuolization and dispersion. The kidneys show an acute exudative nephritis with hemorrhages throughout the cortex.

The tissues of the guinea pig contain many spirochætes, which may be best demonstrated by staining portions of the liver by the older method of Levaditi, making sections and examining by the microscope. Dark field examination of the liver pulp will also usually reveal them.

#### 4. Microscopic examination of the urine for spirochætes.

The urine is centrifugalized and the sediment examined by the dark field method, or films are made and stained by India ink, Romanowsky stain or a silver impregnation method. It will be recalled that the urine contains spirochætes in a variety of conditions, and one must be entirely familiar with the morphology of the *Spirochæta icterohæmorrhagiæ* to hazard a diagnosis by a microscopic examination of the urine.

The microscopic examination of the urine has a special field in expert hands to determine whether a convalescent is excreting the spirochætes in his urine, and is therefore a carrier.

5. Injection of urinary sediment into the peritoneal cavity of a guinea pig.

This method has frequently been followed by positive results, and should be regularly practiced. As in the injection of blood, it has the decided advantage that positive results are well marked, causing the definite pathological changes in the guinea pig, above referred to.

6. Examination of tissues obtained at necropsy by the older method of Levaditi.

By this method the spirochætes may frequently be demonstrated in the viscera, especially in the kidneys.

(b) *In rodents.*

Here, as in the detection of the disease in man, guinea-pig inoculation is the method of choice and reliability. The rats should preferably be taken alive, killed, and the kidney removed at once, with precautions not to contaminate them. The kidneys should then be emulsified and the emulsion injected into the peritoneal cavity of a guinea pig, if possible using a guinea pig for each rat. The guinea pigs should then be observed for at least two weeks. If the *Spirochæta icterohæmorrhagiæ* are present the pig will become ill, show some rather variable pyrexia, become slightly jaundiced, collapse, and die in about 10 days, and at *post-mortem* examination will show the marked picture of jaundice and hemorrhage referred to above. Spirochætes may be demonstrated in the tissues, as previously indicated."

### TREATMENT.

In the absence of a specific chemotherapeutic agent to combat the invading parasites, the treatment should be symptomatic, according to directions mentioned under Acute Catarrhal Jaundice.

### ACUTE CHOLECYSTITIS.

Catarrhal or suppurative inflammation of the gall-bladder may be the result of infection through the common bile-duct or through the blood-stream; gall-stones may be associated or absent. The micro-organisms commonly found are the colon bacillus, typhoid bacillus, staphylococcus, streptococcus,

and pneumococcus. The preliminary symptoms are those of indigestion, followed by pain over the gall-bladder area, with extension of the pain to adjacent parts as the disease progresses. Nausea and vomiting are common and troublesome symptoms. Fever is present when infection is caused by the streptococcus, staphylococcus, and may be absent in colon bacillus infection. The pulse is rapid and small, the respiration is rapid, and conforms to the costal type, and the abdomen is more or less rigid. The patient appears quite prostrated. Palpation reveals tenderness over the gall-bladder, and sometimes a pear-shaped bulging mass may be outlined. The leucocyte count may be normal and below normal in bacillus colon infections, but it tends to become high when the fever is marked. This condition must be differentiated from appendicitis occurring high in the abdomen and from acute intestinal obstruction, in which cases pain is much more severe, and comes on suddenly, with signs of more or less general peritonitis. Affections of the gall-bladder commonly give symptoms referable to its intimate anatomy.

#### TREATMENT.

The patient should be placed in bed, and, if the pain is severe, given a hypodermic injection of  $\frac{1}{4}$  grain (0.01620 Gm.) morphin. Cases of acute cholecystitis of catarrhal type may be treated precisely as catarrhal cholecystitis is managed (*q.v.*). The stomach should be emptied by administering large draughts of lukewarm water and by tickling the palate with the finger. This simple measure is nearly always successful, and should be repeated until the stomach contents are completely rejected. Feeding should be withheld until the patient is more at ease, when hot lemonade or weak tea with crackers or toast may be given. To this may be added strained barley soup, plain broths, and dry toast. The diet should be gradually increased as the symptoms subside. To aid the abatement of the catarrhal symptoms urotropin, 5 grains (0.324 Gm.) three times a day, or sodium salicylate, 10 grains (0.650 Gm.) three times a day, are advocated. If the vomiting or retching is severe, 5 drops (0.30 mil) of chloroform on cracked ice is valuable; or 5 drops (0.30 mil) each of spirits of chloroform and spirits of camphor on ice should be given.



The dietetic treatment should be continued for several weeks after tenderness of the gall-bladder area has subsided.

Repeated attacks of cholecystitis precede gall-stone inflammation. It is for this reason that patients suffering from recurring attacks of pain in the gall-bladder area should prolong their treatment, and to avoid recurrence should maintain a strict dietary. The bowels should be regulated by an occasional saline purge, by the constant use of mineral oil, and by enema whenever the occasion may demand it.

### GALL-STONES.

Inflammation of the gall-bladder or bile-ducts may result in the precipitation of the inflammatory products which when accumulated become organized into hard masses of various shapes and sizes, commonly called gall-stones. These may be found in the gall-bladder, in the cystic or common bile-duct, or deep in the structure of the liver. Cholecystitis is usually associated with or may precede the formation of gall-stones. The typhoid bacillus and the colon bacillus are chiefly responsible for the inflammations due to the precipitation of the stones. Among the predisposing causes are obesity, middle age, female sex, sedentary habits, excesses in fat and starches, chronic constipation, habitual tight lacing, pregnancy, and pancreatic disorders. It may occur in adults, and even in childhood. In many instances there is a history of antecedent typhoid fever.

Stones vary in size from a grain of sand to that of an egg, while the color ranges from light yellow to dark green. They are largely made up of cholestrin, bilirubin, salts of calcium, potassium and sodium, and sometimes traces of iron. They may be round, oval, faceted, smooth or regular, cuboid, cylindrical, hard, or soft.

Gall-stones do not always produce symptoms. Their presence is indicated only when their passage from the gall-bladder through the ducts is obstructed or when associated with an acute or chronic cholecystitis.

A stone impacted in the bile-ducts gives rise to *symptoms* of obstruction and irritation. There is excruciating pain in the right hypochondriac area, oftentimes referred to the right shoulder or directly backwards toward the spine. The pa-

tient appears exhausted, there is quickening of the pulse, with profuse sweating and vomiting. On some occasions the patient will shriek out in pain, following which there is syncope and extreme prostration. There may or may not be any prodroma preceding the attack. Fever may be present or absent, but when associated with gall-stone colic it ranges from 101° to 102° F. (38.3° to 38.8° C.). If the common bile-duct is occluded, jaundice becomes a conspicuous symptom. Cholelithiasis, however, is associated with jaundice in only about 50 per cent. of the cases. Stones may also be passed without any symptoms of pressure or obstruction.

The area of the pain is situated at a point varying from about 3 or 4 inches (7.6 or 10.1 cm.) below the xiphoid cartilage, and about the same distance to the right of the median line. If the cystic duct is obstructed, a distant or bulging mass appears in the gall-bladder area, which is the distended gall-bladder. In cases of obstructive jaundice, the hepatic area may be enlarged for a distance of several inches below the costal border. The areas of the liver and gall-bladder are sensitive on pressure. Following the subsidence of the jaundice and after the passage of the stone, the enlarged hepatic area recedes to normal. The attacks of colic tend to recur, since stones are usually movable. If there is but one attack, it is reasonable to suppose that there has been but one stone of such size as to cause symptoms. Gall-stone colic may last from a few hours to several days, and the pain in some instances several weeks. Urinalysis reveals bile, which may precede the occurrence of jaundice. When the stones are small, the hepatic colic is of mild type, causing a feeling of discomfort and distress. The diagnosis of gall-stones is often very difficult. Symptoms of indigestion, and occasionally pain over the gall-bladder and liver area may lead to suspicion. When the stones become impacted, however, the diagnosis is readily confirmed. The bile in the urine is a warning signal. Stones passing into the intestinal tract may or may not be detected in the feces.

#### TREATMENT.

The most valuable remedy is morphin used hypodermically, in doses of  $\frac{1}{4}$  grain (0.01620 Gm.), repeated in a half-hour if necessary. It is better to use this amount of the narcotic at

first, and to increase if necessary, rather than to give an initial large dose. As an accessory, chloroform may be inhaled until the effects of the morphin are manifest. Among other remedies may be mentioned paregoric in teaspoonful doses (3.75 mils), repeated two or three times, or 10 minims (0.60 Gm.) of deodorized tincture of opium. These remedies, however, taken by mouth may not be readily absorbed, and are not infrequently rejected in the vomitus. Morphin used hypodermically is the remedy *par excellence*, and there is no contraindication to its use in severe gall-stone colic. Hot stūpes may be applied over the gall-bladder area with great relief: hot flax-seed poultices, the hot-water bottle, or the electric pad. After the effect of the morphin has subsided, the patient may experience considerable pain, in which instance paregoric may be given in teaspoonful (3.75 mils) doses every two hours. A valuable prescription consists of sodium salicylate, 5 grains (0.324 Gm.), and codein phosphate,  $\frac{1}{8}$  grain (0.00810 Gm.), every three hours.

Following the attack the patient will naturally have little or no appetite. This is a distinct advantage, inasmuch as all solid food should be prohibited. Hot lemonade or hot tea is comforting. After the patient has had twenty-four hours rest in bed without food, the bowels should be moved by an enema of soap suds or normal salt solution. Strong cathartics are contraindicated, inasmuch as they may precipitate another attack. Enemas when given too early or given carelessly also may be detrimental for the same reason. When the patient is more at ease, broths free of fat are permitted, and plain toast. Cereals with milk are permitted, among which are corn flakes, grape nuts, puffed rice, and others. The light diet should be continued for several days. All fatty foods are to be omitted.

### CHRONIC CHOLANGITIS AND CHRONIC CHOLELITHIASIS.

Stones lodged in the gall-bladder or in the ducts set up a chronic catarrhal inflammation of the invaded areas attended with a group of symptoms depending upon the location of the calculi. Repeated attacks of pain over the gall-bladder area, attended with nausea, vomiting, tenderness, and gastro-intes-

tinal disorders indicate that the gall-bladder and the hepatic and common bile-ducts are the seat of pathologic changes. The gall-bladder may be enlarged and tender as the result of an accumulation of mucus, bile or stones. When, however, the common or hepatic ducts are affected, a catarrhal inflammation is set up which causes more or less obstructive symptoms. Impaction in the hepatic duct causes a general cholangitis attended with exacerbations of acute catarrhal jaundice. Obstruction of the common duct, however, causes enlargement of the gall-bladder and liver attended with jaundice, which is constant and intense, or, if the stone be movable, intermittent. The ball-valve action of the stone is responsible for the changing jaundice and the febrile elevations and recessions. Intense itching is an annoying symptom. Pain, referred to the right shoulder or to the back is paroxysmal, accompanied by chills, fever and sweat. During the interval of attacks, the temperature is normal. Infection by pathogenic micro-organisms may occur during the course of chronic cholangitis and chronic cholelithiasis, causing suppuration of the gall-bladder or a suppurative cholangitis. The characteristic symptoms—chills, fever, and sweat—will be added to those already mentioned. Delay in surgical procedure may bring on a fatal septicemia.

#### TREATMENT.

The treatment of gall-stones is surgical. During the attacks of colic the medical measures already described are indispensable. Between the attacks, however, attempts should be made to abate the catarrhal inflammation of the liver and ducts and to encourage a return of normal function. Dietetic treatment in this respect is the determining factor in aborting future attacks. The acute exacerbations common in gall-stones call for light diet—soups strained of fat, milk, buttermilk, skim milk, toast, crackers, hot lemonade, and tea. It is generally recommended that acid food be avoided. While this is true of tomatoes, vinegar, salad dressing, pickles, and other spiced foods, the juice of sweet oranges used with shaved ice is quite palatable and harmless. Fatty foods, however, are forbidden. During the intervals between attacks all fried foods should be stricken from the menu, but boiled soup

meats, chicken, and tender veal are permitted. Custards, junket, tapioca pudding, floating island, and vanilla ice cream all are to be allowed.

Among the vegetables permitted are peas, beans, asparagus tips, a small amount of butter, spinach, and carrots. Patients troubled with hyperchlorhydria should drink large quantities of water, either pure or containing alkalies, with a view to neutralizing the excess acids. Effervescent phosphate of soda, 1 teaspoonful (3.9 Gm.) in half a glass of hot water before breakfast or given three times a day, tends to stimulate the flow of bile, cleanses the stomach of retained mucus and food and renders the bowel movements soft and effective. Among the natural waters recommended are Carlsbad, Vichy, Neuenahr, and Bedford. Vettmann<sup>51</sup> recommends the following formula:

Magnesii sulph. ....	℥ij	60.0.
Sodii sulphatis .....	℥j	30.0.
Sodii bicarbonates .....	℥iiss	10.0.
M. S.: One teaspoonful (3.75 mls) in glass of hot water, one-half hour before breakfast and one-half hour before dinner and supper.		

Too great emphasis cannot be placed upon the value of rest in the treatment of chronic cholecystitis and cholelithiasis. During the attacks patients should remain in bed as long as there is tenderness over the liver area. Unusual physical activity may precipitate an attack. It is, therefore, advisable that patients during normal intervals should avoid active athletics.

Much has been said regarding the value of treatment at Carlsbad, where good results have been attributed to the routine measures, to the change of scene, and to careful discipline under the supervision of experts. It is believed, however, that just as good results could be obtained at home in the many resorts throughout the United States. Southern California is a most suitable place for sojourn, and the equitable climate is most desirable. If just as much publicity were given to resorts in the United States as have been given to those abroad, it is believed that just as good or even better results could be obtained.

During the attacks of pain, local application of hot Epsom



salt stupes or hot flaxseed poultices over the gall-bladder area give much relief. When the temperature is high, it is often advisable to alternate the hot stupe with cold, especially during the warm months when cold is best tolerated. An enlarged gall-bladder and liver may recede under constant application of hot compresses over the upper half of the abdomen continued at least for twenty-four hours. Colonic irrigation with salt solution is sometimes advisable during the exacerbations. Continued and exaggerated symptoms, however, call for operative interference.

Cholelithiasis considered alone is a surgical disease. Many cases may incidentally get well under persistent medical care and supervision, but with our advanced methods of surgery, asepsis and expert nursing, the removal of gall-stones and the drainage of the gall-bladder during the early stage of the disease promises prompt relief and freedom from complications.

Attempts to remove or dissolve stones by the internal administration of drugs have proved futile. The use of strong purgatives to expel the stones is also without avail. During the intervals of attacks, good results may be obtained by ordering a prescription composed of ox-gall, 1 grain (0.065 Gm.); sodium salicylate and sodium succinate, each 2 grains (0.130 Gm.), in capsule, three times a day. These drugs tend to defer the attacks and lessen their frequency. They do not, however, remove the causative agents, and sooner or later the attacks become so annoying and disappointing to the patient that surgical intervention is indicated. Olive oil has its value in relieving the gastric symptoms, for its use lubricates the bowel and softens the stools. It does not, however, remove predisposing causes or precipitating agents of gall-stones.

When there is continuous loss of weight, loss of appetite, and general physical and mental depression, medical treatment does little or no good. Continued attacks of gall-stones bring about chronic changes in the liver and stomach and seriously interfere with the patient's activities of life, and in such instances, after medical treatment for two or three months without marked improvement, operation should be advised. Much confusion exists, however, among surgeons and internists as to the best time for operation. Some urge immediate interference, while others prescribe a lengthy medical course, re-

sorting to surgical means only when urgent indications arise. Each case, however, presents its own problem, and the decision rests with the circumstances surrounding the patient and the facilities for carrying out the full details of medical care.

When the gall-bladder becomes acutely inflamed and there is pain, fever, and rigidity extending beyond the gall-bladder area, with threatened general peritonitis, or should the symptoms suggest suppuration, surgical interference is of course apparent. Chronic obstruction of the common duct with septic complications also calls for surgical measures. Among other complications demanding the surgeon's attention are perforation and gangrene of the gall-bladder, and hydrops and adhesions causing indefinite symptoms.

Medical treatment alone should be limited to simple catarrhal cholecystitis, cholelithiasis with mild infrequent attacks of colic or without marked physical signs, and to cases which respond readily to common therapeutic measures.

### CIRRHOSIS OF THE LIVER.

#### (Sclerosis of the Liver; Interstitial Hepatitis.)

The term cirrhosis of the liver is commonly used to indicate a chronic disease of this organ characterized by the deposit of an excess of connective tissue. Irritation of the liver continued over a great length of time, regardless of the agency or source, is accompanied by multiplication of the round cell tissue at the expense of the liver cells, encroaching upon the blood-vessels and bile-ducts in such a way as to obstruct their normal circulation, as the result of which there is produced a series of symptoms, both local and general.

Three types of cirrhosis are generally recognized—atrophic, hypertrophic, and biliary. From clinical evidence, however, there is need to mention but two main types, of which there may be gradations having characteristics of both. These clinical types are:

1. Portal cirrhosis, characterized by the excess formation of connective tissue around the portal veins in such a manner as to restrict their circulation, damming back the blood into the spleen, stomach and intestines.

2. Biliary cirrhosis, characterized by inflammatory changes

and thickening of the walls of the bile-ducts, causing the bile circulation to become impeded.

Of the *portal* variety, two forms may be described—*atrophic*, or Laennec's, and *hypertrophic*, or Hanot's cirrhosis. The former is the more common. The liver is, in the early stage of Laennec's cirrhosis, enlarged or normal in size, but later it becomes much smaller than normal. The capsule is thickened, presenting a granular appearance under the surface. The liver is firm to touch and altered in shape. On cutting, it resists the knife. The surface of the skin exhibits a hobnailed appearance, caused by the projection of yellowish areas of liver cells surrounded by grayish white bands of connective tissue. Microscopically, there is an increase of connective tissue about the portal veins and a crowding together of liver cells.

In contrast to this hypertrophic cirrhosis exhibits its connective tissue change in the peripheral zones of the acini extending into the intralobular connective tissue, constricting and obstructing the biliary passages. The liver is enlarged, extending several fingers' breadth below the costal border. It presents a mottled yellowish-green appearance.

Mayo<sup>52</sup> claims that he has never seen a case of Hanot's cirrhosis. He believes that this type of cirrhosis has no pathologic basis and little clinical evidence to support its existence. He contends that a large majority of the cases that have been called hypertrophic or Hanot's cirrhosis are either hemolytic icterus or the ordinary type of biliary cirrhosis. While typical biliary cirrhosis and typical portal cirrhosis exist and are well defined as such, it is easy to deduct that atypical or mixed cases are found, and that connective tissue may be in excess locally or throughout the liver.

*Biliary* cirrhosis results from chronic stasis of bile in the ducts about the bile-ducts, the walls of which are thickened and the lumina constricted; microscopically, it resembles the hypertrophic variety. The walls, however, are more deeply bile-stained. The characteristic changes consist of spots of necrosis in the peripheral zones of the acini. Later these areas are replaced by new connective tissue, and new bile-ducts appear in the intralobular spaces.

Local areas of cirrhosis may occur in tuberculosis, cancer, and syphilis.

Both portal and biliary cirrhosis may be associated with an enlarged spleen. In 51 cases of splenic anemia reported by Wm. J. Mayo, in which the enlarged spleen was removed, the relief to the portal circulation was immediate. The portal obstruction and ascites disappeared. The evidence here points to the fact that the poisonous products were carried to the liver from the spleen, their nature probably being that of a protein derivative filtered from the blood.

Any irritant which reaches the liver from the portal or hepatic circulation or through the bile-ducts may, after acting for a sufficient length of time, result in a reactive stimulation of the connective tissue framework of the liver. Alcohol is the most frequent cause of fibrosis in portal cirrhosis. The degree of connective tissue change is in direct proportion to the amount and strength of the liquors taken. Highly spiced food may also influence the excessive production of fibrous tissue. In cases of chronic autointoxication and in chronic digestive disorders, cirrhosis of the liver may be the resultant factor. Syphilis (especially congenital), diabetes, gout, cancer, tuberculosis, and chronic forms of malaria may be accompanied by interstitial thickening of the liver. Cardiac diseases with insufficiency of the circulation are found associated with secondary cirrhosis. Splenic diseases may also be accompanied by changes in the liver.

Biliary cirrhosis is usually associated with gall-stones, more especially when there is chronic obstruction in the common bile-duct. Jaundice is a common feature. The removal of the stones or drainage of the gall-bladder does not always effect a cure, since some concretions may persist in the small bile-ducts. The damage caused by the stones has already been done, and thickening of the ducts has occurred at the expense of their lumena.

Another type of biliary cirrhosis may result from infection of the gall-ducts and gall-bladder with the streptococcus and colon bacillus. Chronic infection of the biliary passages may be complicated by a chronic pancreatitis.

In hypertrophic cirrhosis there is frequently an absence of definite cause. It may occur in children and in young adults following infectious diseases. It is likely that this disease has often been confused with biliary cirrhosis.

The disease may exist for years without any special *symptoms* referable to the liver. When, however, the portal circulation becomes impeded, a chain of symptoms limited to the abdomen arise. Among the early signs are anorexia, loss of appetite, loss of weight, and general gastro-intestinal disorders such as constipation, indigestion, and jaundice. As the portal circulation becomes obstructed, the patient complains of morning nausea and sometimes vomiting of blood. The stools also may contain evidence of slight bowel hemorrhages. A sense of fullness and pain may be complained of over the hepatic area. As the obstruction increases, it causes the veins over the surface of the abdomen to become enlarged and prominent. This is due to the compensatory circulation established between the internal mammary and the superficial epigastric veins. Loss of weight progresses as the degree of obstruction increases; the cheeks become hollow and the complexion is sallow; the face has a pinched expression. Ascites gradually develops, distending the abdomen, the walls of which become taut and the skin shiny. General symptoms of toxemia soon develop, accompanied by delirium, convulsions, and later by coma. Secondary anemia usually develops, sometimes to an extreme degree. The disease is afebrile, although occasionally the temperature ranges as high as from 100° to 102° F. (37.7° to 38.8° C.). Urine analysis shows an increased specific gravity, a diminished amount of urea, occasionally the presence of bile, and in cases associated with nephritis a variable quantity of albumin and tube casts.

On physical examination the abdomen is found to be distended, the veins prominent, and the skin of a yellowish tinge. The distention oftentimes makes it impossible to outline the lower border of the liver, but after paracentesis its nodular edge can be felt at the costal border. When outlining the liver by percussion, its vertical enlargement, which is normally about 4 inches (10.1 cm.) in length may be either diminished or increased. This variation in size is accounted for by the fact that the early stages of atrophic cirrhosis may be accompanied by the preliminary hypertrophy.

The liver in hypertrophic cirrhosis is enlarged, as detected by percussion. As to the symptoms, jaundice, accompanied by gastro-intestinal disorders, is constant. There may, however,



be an enlargement of the liver long before any constitutional symptoms manifest themselves. As the disease progresses the jaundice increases, with varying amount of fever, periodic attacks of pain resembling hepatic colic, hemorrhages into the skin and mucous membrane, and profound prostration. In some instances fever may be entirely absent. Bile is found in the urine, together with albumin and casts. The spleen may be enlarged, and ascites is rare.

The symptoms of *biliary cirrhosis* are those already mentioned under Chronic Obstruction of the Bile-ducts. Jaundice is prominent and much more intense than in the hypertrophic form. Intermittent fever is frequently observed. Gall-stones, stricture of the common bile-duct, and obstruction by tumors or other agencies, will present their respective symptoms in addition to those of biliary cirrhosis.

#### TREATMENT OF CIRRHOSIS OF THE LIVER.

In the early stage efforts should be made to remove the existing causes, as already mentioned under the discussion of the etiology, to relieve the prevailing gastro-intestinal symptoms, and to adopt a routine diet which will relieve the liver of its imperfect functions, and stay the further development of the disease. Alcoholic beverages should be forbidden. Highly spiced and seasoned foods must be avoided, so that salt herring, spices, beef, mustard, pepper, horseradish, Worcestershire, paprika, catsup, and other acrid condiments must be forbidden. For the mucous gastritis which often accompanies the active stage of cirrhosis, a teaspoonful (3.75 Gm.) of effervescent phosphate of soda in a glass of hot water should be taken one-half hour before breakfast. This tends to wash out the accumulated mucus and favors a laxative movement of the bowels. If there is a great deal of gaseous distention, sodium bicarbonate or magnesium oxide should be substituted. Calomel given in small doses for several days and even weeks may be of material assistance. About  $\frac{1}{6}$  grain (0.01080 Gm.) given three times a day exerts a beneficial effect both upon the function of the liver and the intestinal tract. This statement is made in spite of the recent investigation which claims that calomel has really no distinct chologogic effect.

A great deal has been said in favor of treating chronic liver complaints in places distant from the patient's home. It is believed, however, that this country offers a sufficient number of resorts, both in the East and in the West, where proper and effective systematic treatment may be procured, and where the desired mental diversion and physical rest may be obtained. The present world-wide conflict, which makes it impossible to obtain treatment in other countries, will prove to our satisfaction that cases of liver complaint will make just as many recoveries from home treatment as in foreign resorts across the Atlantic. The constant use of spring waters has a beneficial effect in washing out the stomach and intestines, reduces the toxemia and stimulates the function of the liver and intestines.

In cases of cirrhosis caused by syphilis, iodids are essential. These can be increased in quantity to the stage of tolerance, being mindful, however, not to cause derangement of the digestion. Even in non-specific cases iodids are recommended. The bowels should be kept well regulated by the occasional use of milk of magnesia, sodium phosphate, or citrate of magnesia.

The diet plays a very important and conspicuous part in the treatment. All fats and most acids are to be avoided. This does not preclude such foods as milk and its derivatives, cream, butter, buttermilk, and skim milk, all of which may be taken with advantage. A milk diet alternated with buttermilk is very desirable. One quart (1 l.) of rich milk and an equal quantity of cream buttermilk should be taken daily. It is an advantage to stop all other feeding for one or two days and place the patient on a milk and buttermilk diet, later adding butter crackers, cereals (cornflakes, cream of wheat, shredded wheat, grapenuts), milk and eggs, egg custard, tapioca pudding, gelatin, junket, and ice cream. The sour fruits such as peaches, plums, and pine-apple tend to exaggerate the gastric disturbances. The juice of sweet oranges taken with cracked ice is often quite acceptable. All sour vegetables should be avoided. Among the meats, boiled beef, stewed chicken, and roast veal may be taken. Pork, bacon, smoked meats, shell-fish, and fat fish are unsuitable.

Not only is the dietary to be continued regularly but con-

tinuously until the gastro-intestinal symptoms are in abeyance, the bowels regular, the urine reduced to its normal specific gravity, the urea contents made normal and the subicteroid complexion disappears.

Portal obstruction attended with ascites calls for a removal of the accumulated fluid and the prevention of its return. When the fluid is slight, purgation by the use of compound jalap powder, 30 grains to 1 dram (1.9 to 3.9 Gms.), repeated to obtain the desired effect, may be sufficient. Salts of magnesium, Glauber's salts, potassium bitartrate, and similar preparations may be used advantageously during the early stage of insufficiency of the portal circulation, being careful not to exhaust the patient. Diuretics are, however, preferable. An effective capsule having a decided diuretic action consists of 1 grain (0.065 Gm.) each of powdered digitalis, powdered squills, calomel, and caffein citrate; to be effective 1 capsule should be administered every three hours. If the respiration and the comfort of the patient is disturbed by the accumulated fluid, it should be removed by tapping. (See p. 969.)

Following aspiration of the fluid in the abdomen, the patient should be placed on a salt-free diet in the effort to avoid recurrence of the ascites. At first the patient should abstain from the use of all solid foods. The diet should be prepared in such a way as to avoid all possible use of salt. Such a salt-free diet should be continued to the point of tolerance, at which time salt may be added in small quantities. A diet of milk, buttermilk and skim milk, given alternately, is advocated in the early stages of cirrhosis. Such a dietary furnishes large quantities of water, which produces diuresis and renders the stools soft and free.

The portal circulation is activated by the daily administration of mineral waters, milk of magnesia, phosphate of soda, or compound jalap powder. Calomel in small doses may be given over a period of several weeks. These drugs tend to keep the abdomen free of excess of fluids.

Surgical measures have been advocated by Talma and Drummond for diverting or short-circuiting the portal circulation by establishing a direct communication between the portal and systemic venous circulation. It remains to be proved, however, whether this procedure is practicable.

The terminal stage of insufficiency of the portal circulation is manifested by toxic symptoms, which are either renal, hepatic or intestinal, or a combination of all. Delirium and coma are the terminal symptoms, and the disease may terminate in a fatal hemorrhage. It should be remembered that persons in apparently good health suddenly may develop a serious hemorrhage from the stomach as the result of portal cirrhosis which had not previously been discovered. Hematemesis may be an early symptom due to congestion and stasis of the venous circulation about the cardiac end of the esophagus.

Hemorrhage from the stomach or bowel demands that the patient be placed in bed in a quiet room. Food is withdrawn entirely and an ice-bag placed upon the abdomen. After the patient is quiet and the hemorrhage has ceased for twenty-four hours, rectal feeding should be commenced. After another day milk may be given by mouth, together with such other liquids as the individual case will permit. The occurrence of hemorrhage in the gastro-intestinal tract renders the prognosis extremely grave. Nephritic complications should be treated along rational lines. When delirium occurs, hot packs combined with the hypodermic use of pilocarpin and the internal administration of cathartics may bring about some relief. When the symptoms of extreme toxemia occur, they are indicative of a fatal termination, in which instance efforts should be made to make the patient as comfortable as possible.

### ABSCESS OF THE LIVER.

The liver may be the seat of single or multiple abscesses as the result of invasion of pathogenic micro-organisms through the portal system, bile-ducts, general blood-stream, and occasionally through the lymphatics. When a single abscess occurs, it usually affects the right lobe, and is either deep-seated or superficial. The abscess wall may be thin or thick, depending upon the severity of the infection and the time during which it has existed. The surrounding area is injected, and there is considerable amount of round-cell infiltration, and more or less new connective tissue. The contents also vary according to the type of infection, its severity and

its duration. It varies from a grayish-white to a reddish-brown color, and its consistency may be either viscid or quite fluid in character. When multiple abscesses occur, the surface of the liver is studded with small elevated yellowish tubercles beneath the capsule. Section of the liver reveals small abscesses throughout its structure. Thrombosis in some of the portal vessels may be found. Infection resulting from gall-stones and obstructed bile gives added evidences of these respective conditions. The hepatic cells are in a state of parenchymatous degeneration and there is general turgescence of the circulation. The abscess cavities are filled with pus, necrotic tissue and bacteria. The latter sometimes may be entirely absent. The smaller blood-vessels may be filled with emboli.

Suppuration of the liver may result from traumatism, the presence of gall-stones, and a secondary infection from gastric ulcer, appendicitis, typhoid fever and dysentery. Infections in other parts of the body may be complicated by abscess of the liver, such as long-standing disease of the bones, injuries of the soft parts, and occasionally following scalp wounds. Liver abscess may also result from parasitic invasion by flukes, ascaris, ankylostoma, tænia and oxyuris. Alcoholism and malaria are predisposing factors.

Tropical abscesses may exist with few or no signs referable to the liver, but are attended by diarrheal disturbances. Suspicion of abscess may always be entertained when ameba are found in stools. Abscess of the liver occurs at ages varying from 7 to 50, but most commonly from 20 to 30. About nine-tenths of them occur among men. The abscesses may be small or large, some 4 centimeters in diameter, and others implicating the whole of the right lobe of the liver, containing a liter or more of pus. The contents of a chronic abscess is a thick viscid reddish-brown foul pus, in some instances containing shreds of liver tissue.

The *symptoms* of abscess are very much like those of suppurations elsewhere, with the added symptoms referable to the liver. There is pain and tenderness over the hepatic area, which becomes exaggerated upon changing posture and on pressure. Multiple abscesses may produce few or no physical signs. A single abscess, however, produces quite characteris-



tic symptoms. There is a bulging of the ribs over the hepatic area, and the overlying skin is reddened and inflamed. A deep-seated abscess is less plainly visible externally. The lower border of the liver extends several fingers' breadth below the costal border. Palpation reveals tenderness and in some instances fluctuation in the midclavicular line. The area of hepatic dullness is increased upward both in the midclavicular line and midaxillary line, and can be traced to the angle of the scapula.

Acute abscesses are attended by a sharp rise in temperature ranging from 103° to 104° F. (39.4° to 40° C.), and may be ushered in by a severe chill. The fever is hectic, resembling tertian and quartan malaria, attended by chills, fevers, and sweats. In chronic cases little or no fever is present.

Large abscess may perforate through the abdominal wall into the pleura cavity, bronchi, pericardium, stomach, intestines, or peritoneal cavity, and produce complicating symptoms accordingly. When rupture takes place into the lungs, there is cough, expectoration of an anchovy sauce-like muco-pus. There are also symptoms of a complicating empyema.

Abscess of the liver produces general symptoms characterized by loss of flesh and strength, gastro-intestinal disorders, constipation alternating with diarrhea, and an icteroid tinge of the skin. The *entameba histolytica* may be found in the stools. Pressure of the abscess upward compresses the lung tissue, causing cough, expectoration, dullness, broncho-vesicular breathing and crepitant râles at the base of the right lung. Splenic enlargement is a common accompanying physical sign. Severe toxic symptoms resulting from infection of the liver may bring about a general septicemia or toxemia manifested by severe headache, backache, and pyrexia, delirium, tremor, stupor, and coma.

#### TREATMENT.

Liver abscess should be treated in the same manner as abscess in other parts of the body. Early and free incision with a view of draining the infectious products is the first indication. There are, however, many medical measures which can be adopted preparatory to surgical procedure, especially in cases of multiple abscess where drainage is not feasible.

The temperature can be reduced by tepid sponging, the application of an ice-bag, and the internal administration of quinin. Hot stupes or a hot-water bottle over the hepatic area may tend to make the patient more comfortable. An acute abscess always should be evacuated as early as possible. This may be performed by aspiration with a trocar and cannula, or by the more radical and more effective measure of incision over the bulging mass. Chronic abscesses, especially those originating from dysentery, may be drained by incision through the upper abdomen or by the transthoracic route. Drainage by cannula and rubber tube is recommended by some, but because of the movements of the liver this method has been deemed objectionable.

Much has been said of late regarding the use of ipecac in the treatment of amebic abscesses. Ludlow<sup>53</sup> refers to his experience at the research department of the Severance Union Medical College, Seoul, Korea, regarding the emetin treatment of tropical abscess of the liver, as follows:

"In 1913 emetin was used in three out of four cases, and was given twice a day in doses of  $\frac{1}{4}$  grain each for a week following operation. Except in one case where ameba were found in the feces, it seemed to be of no special importance in hastening the recovery. In 1914 emetin was used in four out of six patients. This time it was given in  $\frac{1}{2}$ -grain doses twice a day for six days. During this period there was no marked improvement which could be attributed to emetin, but it was continued because others had reported good results. In 1915 it was given in 1-grain doses once daily for a week. During this time emetin was used for periods longer than a week, but no rapid progress was noted after its use." It is believed in the experience of Dr. Ludlow that drainage of the abscess gives better results than when emetin is used alone.

Abscess of tuberculous origin always should be evacuated, and the usual treatment of tuberculosis instituted.

### ACUTE YELLOW ATROPHY OF THE LIVER.

This disease is an acute destructive process of the liver cells manifested by jaundice, hemorrhage, and various nervous phenomena. The liver is much reduced in size and weight,

soft and friable, and of a mahogany-brown or grayish-yellow color. Its cut surface presents areas of yellow atrophy intermingled with red pigmented areas of congestion. There is a distinct degeneration of the liver cells, which are replaced by fat globules, cellular *débris*, and blood pigment. The disease is caused by a toxic agent or agents having a special affinity for the liver and causing a precipitation of fat. The exact etiology is uncertain, but among the predisposing causes are acute alcoholism, puerperal fever, typhoid fever, septic infections, malarial fever, and syphilis. The disease begins with severe headache, backache, nausea, vomiting, and fever, and later on deep jaundice supervenes, together with nervous symptoms—delirium, convulsions, stupor, and coma. The vomiting becomes severe and bloody, and hemorrhage may occur in any part of the intestinal tract, in the lungs and in the skin, causing extreme prostration and shock. The urine is highly colored and contains bile pigments, albumin, and casts. There is tenderness over the area of the liver, exaggerated on pressure. The hepatic outline indicates that this organ is considerably diminished in size.

The *treatment* of acute yellow atrophy of the liver is symptomatic. Vomiting should be controlled by gastric lavage, by the use of cracked ice, and by an ice-pack applied to the abdomen. The elimination of toxic products should be attempted by colonic irrigation. Stimulants are called for, and should be administered freely. No special remedy is known for the disease, which is virtually always fatal.

### FATTY DEGENERATION OF THE LIVER.

This may be considered a mild type of yellow atrophy, its pathologic changes being similar but on a smaller scale, and its symptoms less severe. The liver cells are disintegrated and replaced by fat globules, which tend to obliterate the interlobular framework of the liver, causing it to become friable and soft, shrunken and smaller than normal. Granular *débris*, cholesterin and tyrosin crystals intermingle with the newly formed fatty tissue. The disease occurs among alcoholics, workers in phosphorus, arsenic, and other poisonous products, cachetic diseases such as cancer, tuberculosis, pernicious anemia, and in acute infectious fevers.

The *symptoms* indicative of this process may not be manifest at the onset of the disease. Later, however, there is pain over the liver, jaundice, and gastro-intestinal disorders. The severe type resembles very much acute yellow atrophy.

### TREATMENT.

Persons employed among poisons should be protected from their effects by routine systematic supervision of their health. Employers can do much by rendering the workshop healthful by free ventilation and by rendering poisonous materials less harmful through a system of exhausts. Workers should be required to bathe at least once daily, to wash the hands before eating, to change their underclothing at frequent intervals, and to give special attention to their personal habits. A cathartic taken once weekly is very valuable in keeping the intestinal tract free of poisonous substances. When the disease develops, however, the diet should be well regulated with a view of lessening the burden of the liver by eliminating fats and sweets. Effervescent phosphate of soda should be given once daily. The patient should spend as much time in the open as possible, and a tonic administered where the anemia indicates its use.

### FATTY INFILTRATION OF THE LIVER.

This disease is characterized by a deposit of fat in the liver cells, occurring either locally or generally throughout the liver. The organ is enlarged, sometimes assuming immense proportions, being many times its usual weight. The preponderance of fat gives the liver a light yellow color, its sharp borders become rounded, and the hepatic tissue becomes soft. Its cut surface presents a shiny appearance. When examined microscopically, the protoplasm of the liver cells seems to be pushed aside by the invading fat droplets.

The *symptoms* are those usually found in general obesity, except when it occurs in the course of wasting diseases such as cancer, syphilis, tuberculosis, chronic malaria, when the predominating symptoms of these respective diseases mask those produced by the liver infiltration. Extensive deposit of fat in the liver embarrasses the circulation and respiration.

There is dyspnea, arrhythmia, irregular, feeble pulse, and general excitability. The liver is enlarged, sometimes reaching as low as the umbilicus.

The *treatment* of this condition calls for abstinence from sweets and starches. Cereals, potatoes, candies, jellies, and other sweetened deserts should be rigidly avoided. Rye and bran bread should replace wheat. Bacon, oil dressing, fried and fatty foods should be stricken from the diet. A small proportion of butter, however, is permissible. Boiled meats, veal, chicken and fish (except shad), fresh vegetables, and fruits are permissible. Alcohol, of course, is prohibited. A diet of milk, buttermilk, and skim milk, taken alternately for several weeks, may prove of great value. Regulated and systematized baths, taken under supervision, may accelerate the reduction of the fat deposit in the liver. Exercises both indoor and outdoor assist in reducing accumulated fat. The bowels should be kept free by taking 1 teaspoonful (3.75 Gms.) of effervescent phosphate of soda in a glass of hot water every morning before breakfast.

When fatty infiltration occurs in the course of cachectic diseases, fats and sugars should be reduced in quantity.

### AMYLOID DISEASE OF THE LIVER.

This is a destructive disease of the liver in which the protoplasm of the liver cells is replaced by a lardaceous waxy substance—a coagulated albumin called amyloid. It may occur in the liver alone or as a part of generalized amyloid disease. It is found in persons suffering from tuberculosis, syphilis, cancer, and chronic bone infections. The liver is firm to the touch, enlarged, sometimes quite considerably, and on section presents a grayish-brown, shiny appearance.

The *symptoms* are those of anemia—sallow complexion, loss of weight, and gastro-intestinal disturbance. Diarrhea with mucous discharge may be present. The urine is highly colored, containing albumin and waxy casts; an enlarged spleen is usually associated.

The *treatment* should be aimed at the causative agents—tuberculosis, syphilis, cancer, and bone infections. Tonics, fresh, wholesome food, fresh air, and sunshine are the com-



monplace remedies. Syphilis calls for special treatment, while chronic bone infections demand appropriate surgical measures.

### SYPHILIS OF THE LIVER.

The liver is quite often the seat of syphilitic infection, either congenital or acquired. The former is most often found among infants, and is characterized by diffuse infiltration of connective tissue and round cells. The liver is grayish-yellow, larger than normal, and resists the cutting knife. The connective tissue infiltration may be localized in nodular masses, which later undergo contraction, presenting miliary gummata.

Acquired syphilis of the liver makes itself evident some time after the original lesion—a developmental period ranging from three to twenty years. The organ in this instance shows areas of necrosis surrounded by abundant round-cell infiltration and connective tissue, comprising the so-called gummata. These either break down through solution of tissue and are replaced by scars, or they undergo a fibroid change forming hard nodular masses. When liquefaction of the gummata takes place, the resulting scars dent the surface of the liver, which, altered in shape and size, becomes surrounded by dense and firm capsule. In some instances, fibrous protuberances may be seen and felt under the thickened capsule. The liver at first is enlarged, but as fibrosis proceeds it is contracted and diminished in size. The cut surface shows a preponderance of fibrous tissue; the blood-vessels are thickened and hardened. Miliary fibrous masses may also be seen in congenital syphilis.

Syphilis of the liver in infants is usually associated with other signs of a congenital infection. The liver is quite enlarged. In adults, however, there may be few or no *symptoms* at the onset. As the connective tissue changes take place there are signs of portal obstruction and sometimes jaundice. The patient may complain of pain over the liver, which is exaggerated on pressure. Palpation of the liver reveals nodular protuberances on its surface, and on percussion the organ is found to be enlarged. In the later stage, however, the liver is contracted at its lower edge and cannot be palpated. When ascites is present, the usual signs of fluid in the abdomen prevail.

The *treatment* of syphilis of the liver is that of the tertiary stage of the disease. Iodids are given to the stage of tolerance in conjunction with mercury. Active infection as manifested by blood test indicates the need for intravenous injection of salvarsan (arsphenamin), repeated as the individual case requires. When the disease has so far advanced as to cause distinct fibrosis of the liver, very little can be done by way of medication. Congenital syphilis calls for inunctions by mercury and mixed treatment internally. (See Syphilis, vol. i, p. 76.)

### TUBERCULOSIS OF THE LIVER.

Tuberculosis of the liver may be a part of a general infection, and present itself in the form of miliary tubercles scattered throughout the organ, as solitary tubercles or abscesses, as a tuberculosis cholangitis, or as a tubercular cirrhosis. The pathologic findings are self-explanatory from the respective names. The treatment of tuberculosis of the liver is the same as tuberculosis elsewhere, and is therefore not discussed here. (See vol. i, p. 68, *et seq.*)

### TUMORS OF THE LIVER.

Among the tumors affecting the liver are cancer, sarcoma, angioma, adenoma, and cysts. Cancer is most common after the age of 35, and may be primary or secondary. The latter is more frequent among women, being secondary to carcinoma of the uterus and mammary glands. They may be of hard or soft type, resembling scirrhus or medullary carcinoma in other parts of the body. The etiology and general symptoms are those of cancer elsewhere.

Sarcoma may invade the liver in its various forms, either small round cell, large round cell, or melanotic variety. The last-named may follow sarcoma of the orbit.

Tumors of the liver are manifested by pressure-symptoms which interfere with the portal or biliary circulation and by pressure upon the inferior vena cava and adjacent viscera. When the tumor is secondary to growths in other organs, the symptoms referable to these original seats of origin are asso-

ciated. Jaundice, pain, ascites, fever, and cachexia are the dominating signs of malignant growths in the advanced stages. Symptoms of toxemia precede a fatal termination. The liver is enlarged and nodular. The treatment, of course, is symptomatic.

Cystic conditions of the liver may be treated by appropriate surgical measures of drainage. The *x*-ray is valuable not only in diagnosis, but may also be of assistance in conjunction with other measures in the treatment of neoplasms.

## DISEASES OF THE PANCREAS.

### GENERAL CONSIDERATIONS.

In considering diseases of the pancreas, it becomes necessary to study the urine and the feces, in order to ascertain if the pancreas itself is implicated, or if the pathologic process is outside of that organ. So much confusion has in the past arisen in the study of some of the more salient facts bearing upon this subject, that the following epitome has been gleaned from the many investigations of Prof. J. C. Attix, of Philadelphia.

The secretions of the pancreas carry on an enzymotic action on carbohydrates and fats. If the pancreas is at fault, the feces should be normal in color (because of the normal presence of indol, skatol, etc., coming from the liver). If these products are not present, one would suspect a disease of the liver, bile-ducts, or gall-bladder, rather than the pancreas, *or both may be diseased*. If the inflammation of the pancreas is of such an extent as to interfere with its functions, sugar may be found in the urine, and undigested and unemulsified fats in the feces, although the latter would still have the normal color if the biliary secretions have free entrance to the intestines.

Both bile and steapsin emulsify fats. If the inflammation were sufficiently marked to affect the pancreatic duct and common bile-duct, then there would not be an emulsification of fats by either bile or pancreatic secretion, and the feces would be of a light color. Absence of emulsified fats and clay-colored stools are more likely to be connected with a liver

affection. If the feces are normal in color, with the presence of unemulsified fats, and also the presence of sugar in the urine, it is more likely that the pancreas is at fault. In either case it might be the ducts and not the organs themselves, as in the case of a biliary or pancreatic calculus.

The islands of Langerhans are credited with elaborating a specific enzyme and also an internal secretion which convert sugar and maltose into an assimilable form. Amylopsin converts sugars and starches into monosaccharides, and these are taken up by the portal circulation to the liver, where they are again modified into polysaccharides, glycogen, and similar products, so that when finally delivered to the tissues they are readily converted into heat, energy, support general metabolism, and are in a readily combustible form, the  $C_6H_{12}O_6$  being converted into  $CO_2$  and  $H_2O$ .

In either of these cases, where acute pancreatitis with inflammation is sufficient to prevent the entrance of the pancreatic secretion into the intestines, sugar may be found in the urine, not from lack of pancreatic secretion, but from the fact that the inflammatory process prevents its mingling with the substances on which it ordinarily exerts its enzymotic action.

The Cammidge reaction, which some years since gave promise of being an invaluable aid in the differentiation of pancreatic diabetes from diabetes of other origin, has proved itself fallacious. For the muscle juices, pancreatic secretion, and probably the internal secretions of the islands of Langerhans, the adrenals and the pancreas all play a part in the breaking up of the monosaccharides by converting them into carbon dioxid and water, with the development of heat and energy, without leaving behind an excess of sugar floating free in the blood.

Of course, many of the statements and deductions formulated by medical men and widely published are relative and largely in the abstract; otherwise, medicine would lack the essentials of being a scientific study.

The foregoing facts, therefore, may have, in certain instances, their limitations, but as broad, general statements in physiologic chemistry they represent the present state of our knowledge in this difficult field of endeavor, and are herein incorporated in the text, in the hope of clarifying a number

of medical entities, that by their obscure symptomatology too often baffle the physician in his diagnosis.

### PANCREATIC HEMORRHAGE.

Slight hemorrhages into the pancreas are of little clinical interest. They may be secondary to excessive chronic passive congestion, to acute infectious diseases, or the hemorrhagic diathesis. Most cases occur after the fortieth year of life, and apparently at times without cause. Factors favoring pancreatic hemorrhage are: traumatism, arteriosclerosis, alcoholism, and causes most likely acting in bringing about a cerebral apoplexy.

The only constant *symptoms* are sudden agonizing pains, followed by collapse. The pain in certain cases is only trivial. The severe pain complained of is usually referred to the epigastrium; at other times it is not sharply defined. Severe forms of pancreatic hemorrhage usually prove fatal within twenty-four hours, death being caused by reflex paralysis of the heart, due to some coincident vascular affection or to pressure upon the solar plexus and semilunar ganglion.

*Treatment.* This consists in the relief of pain and in meeting the collapsed condition. Morphin should be given to relieve the pain, and the collapse should be treated in the usual manner, by the application of heat, alcoholic stimulation, by the hypodermic injection of atropin and strychnin, and by the use of digitalis. Since death results from pressure-symptoms upon important nerve structures as previously mentioned, it is suggested that free incisions around the pancreas might relieve this condition, and thus be the means of saving the patient's life.

### ACUTE PANCREATITIS.

This is a lesion of the pancreas in which the hemorrhagic process is in association with an active inflammation. Most of the cases reported have occurred in males past 50 years of age. It is more prone to attack the obese than those of a "lean habit." An especial predisposition to the disease seems to be the effects of a gastroduodenal catarrh, gall-stones, alcoholism, and traumatism. Diseases of the gall-bladder are not



infrequently followed by acute pancreatitis, and Flexner, among others, realizing the dominant part played by bacteria, in cholelithiasis and kindred affections of the gall-duct and bladder, produced experimental inflammations of the pancreas by the injection of various bacteria.

The *symptoms* of the disease arise suddenly, and with great violence. There is deep-seated, agonizing pain in the epigastric region, or between the tip of the xiphoid and the navel. This is soon followed by severe and continuous vomiting. Fever is usually slight or may be absent, although in some cases the febrile rise may register as high as 104° F. (40° C.). Among other more or less common symptoms are dyspnea, rapid, feeble pulse, and constipation, although in some cases diarrhea may be present, with watery stools containing free fat. Tympanites occurs in a majority of the cases, and hic-cough and albuminuria may be noted. The intense pain and profound collapse are either dependent upon a circumscribed peritonitis or to pressure-symptoms upon the solar plexus. The diagnosis of acute pancreatitis is best made by exclusion. Surely, the above narration might apply equally well as part of the symptom-complex of many affections. But, in brief, *when a previously healthy person is suddenly seized with excruciating pain in the upper abdomen, with nausea and vomiting and profound collapse, this disease should always be suspected.*

The affections from which it can be quite clearly defined, but which it would be irrelevant to digress upon in this brief review, are: intestinal obstruction, biliary colic, perforating gastric or duodenal ulcer, and the action of corrosive poisons.

*Treatment.* This is merely palliative and symptomatic. The agonizing pain demands full doses of morphin, and the symptoms of collapse should be treated by external heat, the injection of warm saline solutions, and hypodermics of atropin, strychnin, and diffusable stimulants. The diet should be relatively free from fat, and it has been recommended that portions of pancreas be added to the food to be taken by the patient, in the hope that the food is in this way brought more or less in contact with the pancreatic juices, which are so essential for normal digestion. Diastase has also proven its value, when administered immediately after food is taken; to some extent it supplants the pancreatic juices. Later, in the

course of the disease, a supporting nutritious diet is demanded, with the administration of tonics and stimulants.

### CHRONIC PANCREATITIS.

This may follow attacks of the acute form, but more often, especially in alcoholics, it is the result of a persistent or recurring gastroduodenal catarrh, affecting the pancreatic duct.

Conditions giving rise to occlusion of the common or pancreatic ducts, or both, as evidenced in cases of gall-stones, pancreatic calculi, and tumors, occasion a chronic indurative change in the organ. Ligation of the duct of Wirsung in the lower animals is followed by an increase in the fibrous structures of the gland; and in his memorable labors in the investigation of pancreatic diseases Fitz declared that "fibrous thickening of the pancreas is even associated with ulcer of the stomach or suprarenal capsule, aneurysm of the aorta or celiac axis, or with disease of the spine."

The *symptoms* are scarcely indicative of chronic pancreatitis, the patient giving the history of a chronic gastric catarrh of long standing, with frequent attacks of diarrhea. Later there is epigastric pain, slight rise of temperature, and great anxiety. As a result of pressure there may be some ascites and, possibly, jaundice. There is usually progressive loss of flesh and strength. The detection of free fat in the dejections without jaundice and the occurrence of glycosuria and lipuria are of great diagnostic value. The recognition of the disease is extremely difficult; it should, however, be suspected in patients who give a history of long-continued chronic gastric catarrh with frequent attacks of diarrhea, loss of flesh and strength, and the detection of free fat in the dejections.

The presence of glycosuria is of great importance in deciding the true nature of the affection. It is safe to infer that the secreting structure of the pancreas is destroyed, or that there is occlusion of the duct, if, upon the administration of salol, its decomposition is not effected in the intestinal canal, and the presence of carbolic acid fails to appear in the urine.

### TREATMENT.

The main object of treatment in chronic pancreatitis is the proper regulation of the patient's diet. All food requiring pancreatic juice for its digestion should be reduced to a minimum; these include the fats and the starches. When the latter are to form part of the dietary, they should be followed fifteen or twenty minutes after the meal by preparations of pancreatin and soda. Malt diastase combined with alkalies should also be tried. The use of carbonated waters are to be recommended, for Becher has found that they increase the pancreatic digestion and its digestive power in dogs. Other symptoms must be treated as they arise.

### PANCREATIC CALCULI.

These concretions are in all probability produced by a catarrhal inflammation and the retention of secretions in the duct of Wirsung. Other causes responsible for this sort of stone are anomalies of the pancreatic secretion and the presence of cysts or some other factor that causes occlusion of the pancreatic duct.

The first *symptom* suggestive of a calculus is pain, without special tenderness, and due probably to the displacement of the calculus. It strongly simulates biliary colic, and occasionally jaundice is present. The radiation of pain along the lower left costal border rather than to the right side, and at times the detection of fat in the stools or of glycosuria, are great aids in the diagnosis. There is progressive loss of flesh and strength, and stools often contain fat acids, muscular fibers, and at times pancreatic concretions. Occasional or permanent glycosuria may be present.

### TREATMENT.

For the relief of the intense pain that accompanies the passage of a calculus morphin should be freely administered subcutaneously, and, if necessary, ether or chloroform may be given. The application of heat to the abdominal wall may also be of considerable service. The treatment of the ultimate results of pancreatic calculi is that of chronic pancreatitis (*q.v.*). After the calculus has lodged in the excretory duct of

the organ, medical treatment is, of course, unavailing, but it is highly probable that recovery in these instances would follow intelligent surgical intervention. The possibility of the successful removal of a calculus before permanent alterations in the pancreas have taken place should never be lost sight of.

### PANCREATIC CYSTS

These are most common between the ages of 20 and 30, and occasionally follow traumatism. They may occur in children, and by some investigators they are believed to be congenital. The commonest cause is obstruction of the pancreatic duct, and this may be due to inflammation of its wall or to inflammation of the pancreas in the immediate vicinity of the duct, to the impaction of calculi, and pressure of tumors. In a remarkable case reported by Durante, a cyst resulted from the occlusion of the duct of Wirsung by a round worm. Parasitic cysts are likewise sometimes the cause. It is believed by some that many cases diagnosed as pancreatic cysts are really inflammatory conditions of the tissues surrounding the organ, with accumulations of inflammatory products.

Pancreatic secretion is absent, and *pressure-symptoms* are always characteristic of the presence of a cyst. There may be absence of pain, or when present it may occur in severe colicky paroxysms, and be referred to the left epigastrium, left hypochondrium, the left shoulder, and, perhaps, the left half of the face. There is frequent vomiting, constipation, or diarrhea, and the patient complains of a feeling of fullness in the epigastrium, which may be exquisitely tender. Emaciation is frequent, albumin, sugar and often blood are found in the urine. On physical examination the cyst is smooth, elastic, and lobulated. Its growth is likely to be slow, frequently remaining small and stationary for a long period of time, when, after a short interval, it may attain a very large size. It frequently transmits aortic pulsations. It is dull on percussion where tympanitic structures such as the stomach and intestines are not superimposed.

Auscultation may reveal a murmur caused by compression of the aorta. As the cyst increases in size, pressure effects bring about a chain of symptoms that adds much suf-

fering to the patient's already distressing condition. As a result of this mechanical disturbance, atrophy of the pancreas may occur, although digestion and assimilation are not interfered with. Dyspnea, ascites, and dropsy of the lower half of the body are all likely to be in evidence. If the gland is completely damaged, or if the duct is so occluded that pancreatic juice cannot be discharged, the feces may contain fat, and glycosuria may be present, with a decreased amount of indican in the urine. Salivation is sometimes noted.

*Treatment.* There is no medical treatment of pancreatic cysts. When producing permanent or serious discomfort, they call for surgical intervention. The prognosis is good under ordinary conditions, unless diabetes coexists.

## CARCINOMA OF THE PANCREAS.

Benign tumors of the pancreas are rare, and of no clinical interest. Carcinoma of the pancreas is most common in the male sex past the fortieth year, although cases have been reported in the newborn.

Cancer of the pancreas may exist without giving rise to *symptoms*, which when present are referred to gastro-intestinal disturbances. The patient complains of a stubborn dyspepsia, progressive loss of flesh and strength, and gnawing or sometimes sharp epigastric pain. Nocturnal paroxysms of pain are frequent, and are often accompanied with symptoms of collapse. Constipation or diarrhea may prevail, and when vomiting occurs blood or free fat may be found in the ejected matter at times. Blood may be passed by the bowel, but the presence of fat or fat acids are rarely demonstrable; the stools are light in color, and when diarrhea is not present much undigested muscular fiber is found in the dejections. The urine may be increased in quantity, and sugar and albumin are sometimes present. The characteristic feature of cancer of the pancreas is the discovery of a palpable tumor in the region of the gland. It is deep-seated in the middle line and above the navel. It is very slightly movable, and varies greatly in contour, shape and density, and usually transmits the pulsations of the abdominal aorta. The pressure-symp-



toms are identical with the mechanical disturbances produced by pancreatic cysts (*v.s.*).

### TREATMENT.

The symptoms of pancreatic cancer that require treatment are pain and ascites. The pain is to be relieved by hypodermic injections of morphin, and the ascites, if the cause of much distress, should be treated by abdominal paracentesis. (See p. 969.) Of course, all other treatment must be of a symptomatic kind. Diastase and portions of raw pancreas may be given with the food, with the hope of maintaining the digestive functions. In the very early stages, if the disease is recognized, an operation may be the means of saving life, and, according to Koerte, recovery has followed in several cases the extirpation of the tumor, but as a rule this malignant neoplasm is so insidious that when diagnosed it is too far advanced for an operation.

### BIBLIOGRAPHY.

1. Daland, J.: Pa. Med. Jour., 1917, p. 771.
2. Freudenberg, H.: Lancet-Clinic, 1916.
3. Price, W. A.: Jour. Amer. Med. Assoc., 1917, 424.
4. Rhein, M. L.: *Loc. cit.*, 417.
5. Talbot, E. S.: *Loc. cit.*, 420.
6. Potter, H. E.: *Loc. cit.*
7. Anders, J. M.: Principles of Medicine, Phila., 1909, 734.
8. Sippy, B. W.: Therapeutics of Internal Diseases, Forcheimer, 1914, iii, 65.
9. MacCarty and Wilson: Am. Jour. Med. Sc., Dec., 1909.
10. Lyon: A Study of Gastric Sediments and Their Interpretation. Am. Jour. Med. Sc., Sept., 1915.
11. Fenwick: British Med. Jour., 1909, i, 1297.
12. Smithies and Ochsner: Cancer of the Stomach, 1916, p. 440.
13. Whiting: Annals of Surgery, May, 1916.
14. Bolton: Ulcer of the Stomach, 1913, p. 315.
15. Musser and Kelly: Practical Treatment, iii, 348.
16. Lockwood: Diseases of the Stomach, 1913, p. 183.
17. *Loc. cit.*, 176.
18. Smithies and Ochsner: Cancer of the Stomach, 1916.
19. Fenwick: Cancer and Other Tumors of the Stomach, Phila., 1903.
20. Cabot: Jour. A. M. A., Dec. 28, 1912, p. 2395.
21. Hoffman: Trans. A. Gynec. Soc., 1913; Surg. Gynec. Obst., 1914, p. 726.

22. Febiger: Berl. klin. Wochnschr., Feb. 17, 1913, p. 289.
23. Taschenbuch der Magen und Darm Krankheiten, 1912.
24. Lyon: Study of Gastric Sediments and Their Interpretation. Am. Jour. Med. Sc., 1915, p. 402.
25. Fuld and Levinson: Biochem. Ztschrft., Berl., 1907, Bd. vi, Hft. 5 and 6, p. 473.
26. Schryver and Singer: Quarterly Jour. Med., London, Oct., 1912, p. 71.
27. Sorenson and Schiff: Ztschrft. f. physiol. chem., 1909, xiii, 27.
28. Neubauer and Fisher: Deutsch. Arch. f. klin. Med., 1909, xciii, 499.
29. Weinstein: Jour. Am. Med. Assoc., 1910, lv, 1085.
30. Gossett: La Presse Med., Mar. 16, 1912.
31. Smithies and Ochsner: Cancer of the Stomach, 1916.
32. Fenwick: Cancer and Other Tumors of the Stomach, Phila., 1903.
33. Lockwood: Diseases of the Stomach, 1913, p. 277.
34. Brooks: Med. News, May 14, 1898.
35. Salomon: Trans. Path. Soc., London, 1914, lv.
36. Coley: Annals of Surgery, Sept., 1891.
37. Chiari: Festschrift für Rudolph Virchow, 1891, ii, 297.
38. Morgan: Am. Jour. Med. Sc., Mar., 1915, cxlix, No. 3, 392.
39. Lyon and Eiman: Am. Jour. Med. Sc., Dec., 1914, No. 6, cxlviii, 885.
40. Gradwohl: Medical Fortnightly, Oct. 26, 1914.
41. Lyon and Eiman: *Ibid.*
42. Bassler: Diseases of the Stomach and Upper Alimentary Tract, p. 679.
43. Lockwood: Diseases of the Stomach, p. 298.
44. Mohler and Funk: Am. Jour. Med. Sc., Sept., 1916, No. 3, clii, 355.
45. Riviere and Morland: Tuberculin Treatment, Oxford Med. Pub.
46. Holt: Jour. Am. Med. Assoc., 1917, lxviii, 1517.
47. Koplik: Am. Jour. Med. Sc., 1908.
48. Plummer: Cardiospasm. St. Mary's Hospital, Rochester, Minn., Collected Papers, 1911, i, 33-49.
49. Abrams' Spondylotherapy, Philopolis Press, San Francisco.
50. Abrams and Jarvis: Arch. of Diagnosis, 1912, 329.
51. Lyon médicale, 1885. Revue de Médecine, 1887, p. 75. Enteroptose et Neurasthénie, Soc. Méd., des Hôpitaux, Paris, 1886.
52. Vettmann, Henry Wald.: Forcheimer's Therapeutics of Internal Diseases—Billings, 1917, iii, p. 272.
53. Mayo, Wm. J.: Jour. Am. Med. Assn., May 11, 1918, p. 1361.
54. Ludlow, A. I.: China Medical Journal, May, 1917.



# Diseases of the Peritoneum

BY

WILMER KRUSEN, M.D., LL.D., F.A.C.S.,

Professor of Gynecology, Temple University of Philadelphia, Department of Medicine; Chief Gynecologist, Samaritan Hospital; Gynecologist, Garretson Hospital; Director, Department of Public Health and Charities, Philadelphia.





# Diseases of the Peritoneum.

---

## FOREWORD.

THE treatment of peritonitis, as described in this section, has been considered from the standpoint of the general practitioner, and not from the viewpoint of the specialist. The physiology and pathology of the peritoneum have been reviewed only in so far as they may assist the physician in obtaining a clear and comprehensive understanding of the principles underlying the rational treatment of the peritoneal diseases.

Throughout this article it has been repeatedly emphasized that peritonitis is practically always associated with organic diseases, and that the treatment must necessarily be considered in conjunction with the primary lesions in those structures which are anatomically and physiologically related to the lining of the peritoneal cavity.

Each case must be treated on its own findings, giving due consideration to the type of patient and to the resistance to the variety of infection present.

The most modern and accepted methods of treatment have been described as practised by the author and others most qualified to speak authoritatively on the subject.

## ACUTE GENERAL PERITONITIS.

*General Considerations.* In order that one may fully appreciate the rational treatment of peritonitis of all types, it is essential to know the basic facts regarding the anatomic structures and the principles of the physiology of the peritoneum. This structure is the lining membrane of the abdominal cavity, and is classified among the serous tissues, being occluded from the outside of the body. In the case of the female, however, there is a direct communication through the fallopian tubes. This fact should be borne in mind when considering pelvic peritonitis, which in many instances has its origin by way of the genital tract.

The peritoneal cavity, which is formed by the folding and dipping of this serous membrane, is divided anatomically into two cavities, called the greater and lesser sacs, the former being that portion anterior to the stomach and liver wall, and the latter lying directly behind these same organs. The sacs communicate directly with one another through the foramen of Winslow. Inflammatory conditions in one sac may, therefore, spread to the other. That portion of the peritoneum which covers or partly covers the various abdominal viscera is known as visceral peritoneum, while that which lines the abdominal wall is termed the parietal. This differentiation must be made because inflammatory conditions of either portion present their special symptoms. Where the peritoneum covers an organ, it becomes an intricate part of that viscus, helping to make up the structure, and by strong reduplication holds it in place. This is true of the liver, which is held tightly in place by folds of the peritoneum and divided into lobes by invaginations or dipping down of the peritoneum. These strong, firm bands of tissue derived from the serous lining of the abdomen play an important part when inflammatory conditions arise in the region of the liver, being better able to resist invasion than parts which are thin and less resistant to pressure and inflammation.

The omentum is the largest reduplication of the peritoneum, and acts as a protective covering of the organs behind it. When the abdominal viscera are invaded by disease, this apron of tissue with its supply of fat acts as a bulwark of defense in the attempt to limit inflammatory processes by the extravasation of serum, fibrin, leucocytes, and by matting together the tissues about the affected parts.

*Absorptive Power of the Peritoneum.* The peritoneum is a lymph sac, being capable of excreting and absorbing fluid. It has been described as a large joint, lined with synovial membrane, and containing a serous fluid. Because of its extensive area, its absorptive power is said to be very great, and may increase the body-weight fully 10 per cent. in thirty minutes by the absorption of hypotonic salt solution. Advantage of this function is taken in the treatment of peritonitis by the use of enteroclysis. In the region of the diaphragm and small intestines absorption is greatest. Infectious and toxic material when

permitted to come in contact with these portions of the peritoneum may rapidly invade the blood-stream after passing through the larger lymphatics. These parts of the peritoneum are called the danger zones, since absorption of poisonous materials may give rise to generalized blood-poisoning. For this reason patients are placed in the semi-recumbent position in order to keep the infectious material away from the vital parts of the peritoneum.

*The Secretory Function of the Peritoneum.* An albuminous fluid is secreted by the peritoneum in sufficient amount to facilitate the motility of the various abdominal organs. The omentum is probably the most active part of the peritoneum in this process of secretion, and plays an important part in checking inflammation. Robinson<sup>1</sup> describes it as "a man-of-war ready at a moment's notice to move to invaded parts. It circumscribes abscesses, repairs visceral wounds, and prevents adhesions of movable viscera to the abdominal wall. It is like a moving sentinel, whose beat is the whole peritoneal cavity. It is a diagnostic aid directing the surgeon to the original seat of peritoneal disease, where it first contracted adhesions. It closes intestinal wounds, resists infection by exudates, and does not permit absorption of sepsis. It is a storehouse of fat, and acts as a peritoneal drain."

*Nerve Supply.* Being abundantly supplied with nerves, which regulate the caliber of the blood-vessels, and hence the blood supply, the peritoneum is very susceptible to shock. Severe prostration in acute peritonitis may be explained by the intimate supply of sympathetic nerves, causing symptoms in organs distant from the seat of trouble. The visceral peritoneum, being more abundantly supplied with nerves than the parietal, is more susceptible to shock. Pain, however, comes chiefly from the parietal peritoneum, which is supplied by sensory nerves, branches of the ilio-lumbar and the last two thoracic nerves. The visceral peritoneum is comparatively free of sensory nerve supply. Irritation in the peritoneal cavity, therefore, must be sufficient to reach the parietal peritoneum in order to cause pain. Inflammatory conditions may take place in the liver, gall-bladder, ureter, spleen, or urinary bladder, and give no pain locally until the parietal peritoneum is affected. When a stone passes through the ureter, however, the impres-

sion is carried by the sympathetic nerve supply to the cerebro-spinal nerves by way of the spinal cord, and the pain is referred to the groin. In the case of gall-stones, pain is usually referred to the right shoulder. This is explained by the fact that the impression caused by the stones is transferred to the diaphragm, which is in close proximity, and supplied by the phrenic nerve, having its root in the fourth cervical segment of the cord. The nerves supplying the right shoulder are also derived from the same root. Pain travels through this course from the gall-bladder to the diaphragm, to the cervical cord, and thence to the shoulder. Inflammatory conditions in the abdomen which do not implicate the parietal peritoneum, therefore, may cause pain in places distant from the seat of trouble.

*Lymphatics of the Peritoneum.* Of no small importance are the lymphatics of the peritoneum. They are very active in the absorptive function of the abdomen, and are most plentiful surrounding the stomach, liver, and pancreas, less numerous about the abdominal aorta, and least in number in the pelvis. Absorption will thus be greatest in the upper abdomen, and least in the pelvis. Advantage is taken by the surgeon of this distribution of the lymphatics by placing the patient suffering from profuse inflammation of the peritoneum in such a position that the toxic products will gravitate to the dependent portions of the abdomen, where little or no absorption takes place. The semi-recumbent posture is, therefore, adopted in septic conditions of the abdomen, this being known as the Fowler position.

*Summary of Peritoneal Functions:*

1. *Absorption*, which takes place by (a) lymphatics; (b) veins, probably through peritoneal pressure, which is greater outside the veins; (c) osmosis, by direct action of the endothelial cells.
2. *Secretion*, which takes place by exudation from the venous capillaries.
3. *Supportive*. (a) Acting as suspensory ligaments for the liver, stomach, and intestines; (b) encapsulating the pancreas, and adding support to the large intestine, bladder, and uterus.

The leading facts of the *clinical pathology* of acute diffuse peritonitis relate to a uniform distention of the intestines.

Some areas being more affected than others, are distended with gas and matted together by exudate. Depending upon the severity of the infection, the exudate may be *serous*, in which the irritant producing the inflammation causes an excessive transudation of peritoneal fluid. When the irritation is still greater, causing precipitation of fibrin in addition to the serum already present, the exudation is then called *serofibrinous*. The proportion of serum and fibrin will vary, and either may predominate. If the inflammatory condition is still more violent, leucocytes are called into play, which migrate through the walls of the blood-vessels and cause the exudate to become milky, creamy, or greenish-yellow, depending upon the presence of various bacteria. This type is known as the *purulent* variety. The amount of pus formation is dependent upon the severity of the irritation, the resistance of the patient, and the type of prevalent micro-organism. The staphylococcus imparts a yellowish or creamy color to the pus, the pyocyaneus gives a greenish color, while the bacillus prodigiosus imparts a red color. Usually there is mixed infection, and the color of the pus depends upon the predominating micro-organism. When bleeding occurs, caused by the rupture of blood-vessels, the blood is intimately mixed with the exudate, which is then termed *hemorrhagic*. Any of the previous types may later become hemorrhagic.

The micro-organisms found in acute general peritonitis may be of many types, the most common being the staphylococcus, streptococcus, colon bacillus, and less frequently the pneumococcus, gonococcus, and bacillus lactis aërogenes. In this connection the common forms only will be described. Mention, however, may be made of the pneumococcic infections, which have received greater attention of late owing to the new classification of this bacterium into four types, as described by Cole, each of which has special characteristics in morphology and virulence. Our frequent winter epidemics of pneumonia, attended with a high mortality rate, may account for many of the infections of the intestinal tract with this micro-organism. Type I. and type II. pneumococci have been found most frequently in acute pulmonary infections, and there is reason to believe that the same type may be found in acute peritoneal infections.



There are no definitely defined *symptoms* of general peritonitis, for the reason that they will vary with the organs affected, the type of micro-organism which predominates, and the resistance of the patient. Children present different symptoms from those observed in the aged; the streptococcus produces a more overwhelming inflammation than the colon bacillus; and, briefly, the symptoms vary according to the organ chiefly damaged, and the weak will present different reactions than the strong.

We must, therefore, look upon acute general peritonitis as the result of the reaction of the peritoneum against invading poisons, some of which have been absorbed by the blood, causing a series of local and general constitutional symptoms.

When the entire peritoneum is inflamed, which is nearly always true of a mixed infection, the intensity of the symptoms will vary with the predominating micro-organism.

The streptococcus causes the most violent type of infection. It produces a large amount of toxin, which is set free and absorbed. The resulting symptoms will necessarily be high fever, ranging from 103° to 105° F. (39.5° to 40.6° C.), depending upon the toxic effect upon the thermogenic centers, and the pulse will be full and bounding. The severe irritation causes abundant serous exudate to be poured out, distention of the abdomen, and induration of the abdominal walls. The intestines are "splinted" and matted down by the exudate, which is Nature's method of protecting the abdominal viscera from irritation. Peristalsis is thereby hindered, resulting in constipation, distention by gas, and a general sense of fullness of the entire abdomen. When this distention becomes so great as to exert pressure upon the stomach, it produces a feeling of nausea, which later leads to vomiting. Continued obstruction of the normal peristaltic movements, with excessive generation of intestinal gases, causes reversed peristalsis and vomiting of ordinary stomach contents, of bile, and even of fecal material.

The stomach normally eliminates poisonous products from the body, just as in the case of morphin poisoning, when this drug can be found in the stomach contents. In general toxemia caused by peritonitis, the stomach also attempts to eliminate the poisonous products, and in so doing the toxins may cause the capillary blood-vessels to rupture, resulting in the extra-

vasation of blood, and accounting for the bloody vomitus in advanced cases of general peritonitis. The dark-brown coffee-ground appearance is due to the action of the gastric juices upon the blood. When the vomitus contains blood, we are assured that we are dealing with a very severe fulminating phlegmonous type of infection, and if further progress of the disease is not arrested immediately, the blood system is overwhelmed with poisons, the antibodies are overcome by the toxins, and we have a condition of general septicemia, which may cause immediate death.

The colon bacillus infection, on the other hand, causes symptoms which are directly opposite to those of the streptococcus. Instead of a violent, rapid and reactive infection, the symptoms are very insidious in onset. The toxins are only mildly irritating, and produce a slow but certain poisoning of the system. The pulse may be slow, of small volume, and low tension. Symptoms of a slowly depressing character may continue over a long period of time, attended by loss of weight, sallow complexion, neurasthenia, and loss of mental and physical activity. These indefinite symptoms may hide the true inflammatory condition, until a slowly forming abscess in any part of the abdomen ruptures and ushers in the acute manifestations of peritonitis. Throughout the inflammatory process a normal or even subnormal temperature may be recorded, and little or no pain complained of in spite of the continued depressing action of the toxins.

The staphylococcus infection gives rise to symptoms midway between those of the streptococcus and the colon bacillus varieties. The fever is not very high, the pulse may be strong or even normal, the pain is slight, and the digestive symptoms merely enough to indicate a mild inflammatory process. There may, however, be several micro-organisms responsible for the peritoneal infection, in which case the predominating germ is manifested by its specific symptoms. This is known as the mixed infection, which is probably the most frequent variety.

Presupposing a typical case of acute general peritonitis, the early cardinal symptoms will be *pain, fever, nausea, constipation, tympanites, and abdominal distention*.

These symptoms correspond exactly with those of typhoid fever, with the exception that in the latter disease the fever

makes its appearance early, and the pain, which is not very severe, late.

*Leucocytosis as a Diagnostic Symptom.* The increase in the number of leucocytes in the blood is the direct result of a violent reaction caused by foreign products in the body. In the early stage of general peritonitis, leucocytosis may not be very marked, but the relatively large percentage of polymorphonuclear cells should be regarded as a significant diagnostic sign of an acute inflammatory process. As the disease progresses, however, an increased number of leucocytes is called into action, when the leucocytosis may reach 40,000 to 50,000, with a preponderance of polymorphonuclear cells, averaging 80 to 90 per cent. This is especially true when the infection of the peritoneum has become generalized and is caused by the streptococcus. A low leucocyte count in a patient who presents weak cardiac sounds, marked physical depression and prostration, feeble pulse, with little or no pain, no distention of the abdomen, and no apparent rigidity of the abdomen, should lead to the suspicion of infection with the colon bacillus. The surgeon must, therefore, be on his guard not to disregard a low leucocyte count or to be misled into believing that no infection is present. On the contrary, the inflammatory process may be general, and the toxins present of such a nature as to be inactive in calling out the leucocytes. In cases of marked collapse, or where the patient is feeble, no leucocytosis is to be expected, and yet the patient may die without any reactive inflammatory signs, without fever and without pain, but by slow and gradual poisoning. The absence of leucocytosis, therefore, may mean a mild inflammation or a severe infection of high toxic nature, with feeble resistance on the part of the patient. A high count, on the other hand, always indicates a severe infection with adequate reaction by the patient. A high count calls for immediate operation; a low count, with signs of severe prostration, calls even more urgently for immediate operative interference.

*Symptoms Following Early Stage.* After forty-eight hours of pain, fever, muscular rigidity, nausea and distention, general symptomatic treatment may cause these symptoms to remit, and the patient may apparently feel relieved. Such relief, however, may be only temporary, and the case may con-

tinue on with cold sweats, pinched facial expression, sunken eyes, sighing respiration, and intense rigidity of the abdomen; the pulse becomes weak, and the temperature may even become subnormal. This group of symptoms should be recognized as those of shock, which is the result of the severe impression made by the general toxemia upon the nervous system. Even in this condition the patient's mind may be clear and the memory good. A similar picture often is seen in the advanced state of cholera, known as the algid stage. For this reason the same name has been applied to the exhausted state in advanced acute general peritonitis. In the colon bacillus infection this algid stage is often presented without any previous violent reactive symptoms.

*Cause of Death in Acute General Peritonitis.* Septicemia is usually the cause of death in general infection of the peritoneum. The invasion of the blood-stream by pathogenic micro-organisms and their toxins is the result either of slow absorption, as in the case of colon bacillus infection, or is rapid and abrupt as in a streptococcus infection. In either variety there is an exhaustion of the antibodies in the blood or an overproduction of toxins when septicemia occurs.

**Subacute General Peritonitis.** When infection takes place in a robust individual, the reaction may not be very violent, and several days may elapse before acute symptoms are manifest. In cases where there is a local peritonitis, or an oozing from a ruptured viscus, the general peritonitis takes place slowly, and the reaction on the part of the patient responds to the steady flow of infection. The symptoms, therefore, correspond in name to those of the acute variety, but will be less severe in character. Moreover, in the case of the slow infecting process more time is allowed for adhesions to form about the inflammatory area, thereby lessening the extent of the inflammation and the severity of the clinical picture.

*Sources of General Peritonitis.* Infection of the peritoneum may arise from causes within the digestive tract, such as a perforated typhoid ulcer, a perforated gastric ulcer, intestinal obstruction and gangrene of the intestines; or from causes outside of the digestive tract, such as perforating wounds of the abdomen, rupture of the urinary or gall-bladder, extension of the inflammatory process from the kidney, pancreas, spleen,



uterus, and from rupture of a localized peritoneal abscess. The common abdominal abscesses are : appendiceal, subphrenic, and pelvic. Extravasation of the intestinal or the gastric contents may cause a general or localized peritonitis, depending upon the resistance of adhesions already formed about the inflamed parts, and the intensity of the infection will depend upon the predominating micro-organism present. In the case of a ruptured appendiceal abscess, encapsulated only by a thin fibrous wall, a severe, violent, and rapidly spreading peritonitis may take place. On the other hand, should this abscess be confined by a tough resisting fibrous wall, which by degenerative process presents a leak, the extravasation of its contents will necessarily be gradual, and the abdomen by its natural process will take care of this infection by newly formed adhesions. The symptoms, therefore, tend to be of mild character and less toxic.

When the pancreas is ruptured as the result of pathologic changes, its chemical secretions are extravasated into the peritoneal cavity, and by their digestive action bring about an acute inflammatory peritonitis.

When obstruction of the bowel occurs, either by invagination of the intestines, twisting of the gut, constriction, or pressure, the blood supply of the parts affected is immediately shut off, resulting in gangrenous degeneration. The intestinal bacteria are then free to migrate through the intestinal walls into the peritoneal cavity, where an acute inflammatory process takes place.

In embolism of the mesenteric artery a change results similar to that just described in obstruction of the bowel. The abdominal viscera supplied by this artery becomes gangrenous, and the invading micro-organisms attack the peritoneal lining.

Inflammatory processes are quite often localized when the affected parts are situated so as to be protected by the suspensory ligaments formed by the reduplication of the peritoneum. This is especially true in the region of the liver, where infection usually results in the formation of a subphrenic abscess.

Acute perforations of the stomach and bowel may become plugged by the adjacent omentum, which checks any spread of intestinal or gastric contents by the exudation of serum and



fibrin, thus localizing the infection and preventing a widespread peritonitis.

### TREATMENT.

Each individual case must be treated according to its own merits. The young and the old, the robust and the weak, will necessarily present different types of cases. Due consideration must be given to the fact that the disease alone is not being treated, but the disease in a particular individual. Every case of general peritonitis has a definite focus of infection. The treatment, therefore, should begin long before the infection has affected the whole peritoneal cavity. In other words, by removing a local infection in the peritoneal cavity, a general inflammatory process may be aborted.

When the entire peritoneum is implicated, however, the first aim in treatment is to "splint" the intestines. This may be accomplished by withholding food and drink of all kinds. In the absence of intestinal contents, peristalsis is necessarily reduced. Cathartics must be avoided, because any movement by the bowel must tend to disseminate the infection in the abdomen and bring about an unfavorable condition. The physician must not be tempted to administer opiates or their derivatives to allay pain or to reduce peristalsis. While the administration of narcotics may relieve the patient, it is only apparent and temporary. Furthermore, narcosis masks the true symptoms, which are the surgeon's indicators as to the time for operative interference. Under the influence of morphin an appendiceal abscess may rupture without giving the usual diagnostic symptoms, and thus permit a rapid dissemination of the infection. It is, therefore, a dangerous procedure to administer narcotics in the treatment of acute general peritonitis. Should the physician decide to adopt surgical interference, however, then, and only then, is it advisable to give a hypodermic injection of morphin. Under its influence the patient is better able to undergo the surgical ordeal, and no danger exists because the abdominal cavity will shortly be explored by the surgeon.

*When to Operate.* If the symptoms become exaggerated after the patient has been resting in bed for twenty-four hours without food and drink, then no time should be lost in opening the abdomen. Should the symptoms become violent and fulminating before the termination of the first twenty-four hours, and

the patient is in severe pain, or in a state of shock, interference should be made at once. Delay even for one hour may prove fatal in violent infections resulting from a ruptured abscess, acute obstruction of the bowel, or rupture of one of the abdominal viscera. Medical treatment is only suggested when the inflammatory process is mild and the symptoms are stationary or are regressing. It is an error to wait for absolutely definite indications. When in doubt, always operate. The danger lies not in the operation, but in the failure to operate. Very often, however, the surgeon receives the case too late for surgical interference. The medical practitioner should always consider the advisability of operative measures first, and medical treatment only as a temporary substitute. Statistics and experience prove that the mortality rate of peritonitis is directly proportional to the time of operation. The earlier the operative measures are adopted the less is the death-rate.

The treatment of acute general peritonitis is surgical. While a few mild cases may be carried along, and even get well, by medical treatment, the adoption of such a procedure is dangerous to life. The time to operate is early, and the earlier the better the prognosis. Never treat a case of general infection more than forty-eight hours by medical means. If no signs of improvement occur, and the infection is violent, the surgeon should not even wait one hour before operating.

*Surgical Treatment.* An incision is made in the median line, the abdomen opened, and a careful examination made for the local seat of trouble. The viscera should be handled gently in order to prevent the spread of infection to other parts and to avoid shock. Inspection should be made of the appendix, stomach, gall-bladder, intestines, and, in the female, of the fallopian tubes. It should be remembered that more than one condition may exist at the same time, and the surgeon must not be content with the finding of one single lesion. The treatment of general peritonitis is, in short, the removal of the cause.

The technic of the various operations will not be discussed here. If there be a perforation of the stomach or intestines, this must be closed; and if the appendix is diseased, it should be removed. An abscess should be opened and drained, and a gangrenous gut must be resected and the healthy segments united.

*To Drain or Not to Drain.* A general infection indicates that the inflammatory process has already extended beyond the local source of the disease. It becomes necessary, therefore, to drain off the toxic products which remain in the peritoneal cavity. If the infection has been a slow one, occurring in a robust patient with good bodily resistance, drainage is not necessary, for the vital powers of the patient may be sufficient to take care of the abdominal infection. Every case of severe and extensive peritonitis calls for drainage. Every infection in persons who are physically weak, and, in the judgment of the surgeon, are unable to take care of the inflammatory process, should be drained. The question of drainage, therefore, rests with the nature of the infection and the type of the patient.

Drainage of the abdomen should be performed (1) if the patient is weak; (2) if there is a fulminating infection present, as shown by the character of the symptoms and the absence of adhesions about the source of inflammation; (3) if there are abscesses in the various abdominal fossæ or between the knuckles of gut; (4) if after removing the cause there is still pus or gangrenous tissue left behind; and (5) when in doubt.

*How to Drain.* Plain sterile gauze may be used to drain the abdominal cavity of its infectious material. This acts as a lamp wick, carrying the septic products from the seat of disease to the outside of the abdomen. The constituents of this inflammatory product soon clog up the interstices of the gauze as the result of fibrin formation, thus hindering further drainage. The gauze drain accomplishes its purpose, however, during the first twenty-four hours before fibrin formation is complete. Failure to remove gauze packing which has become saturated with pus, blood, serum, and fibrin, after twenty-four hours of use, may act as a plug rather than as a wick, and thus hinder the entire purpose of drainage. When packing is used for the purpose of walling off infection, removal is not necessary, because the presence of the gauze is intended to stimulate the formation of new fibrous tissue.

Sufficient gauze drainage should be used to cover all raw surfaces surrounding the local area of infection, and should be long enough to reach beyond the abdominal wound. Protection of the parietal peritoneum and the wound itself may be

accomplished by the use of a rubber dam so wrapped about the gauze as to prevent the contact of the latter with the abdominal wound. Such a drain is sometimes called a cigarette drain. Attention should be given especially to local pockets, where infectious material may find lodgment, as in the case of the iliac fossa. When the infection is severe, and a large amount of pus is found in the abdomen, a counter-puncture may be made in the median line, just above the pubic bone, and a glass tube inserted reaching the cul-de-sac of Douglas.

Another method of draining the abdomen of its toxic products is the continuous instillation of normal saline solution into the bowel, where the fluids are absorbed and eliminated through the open abdominal wound and through the kidneys. This is known as the "Murphy enteroclysis." The method of using this drainage can best be described by repeating the words of Dr. Murphy himself:

"As soon as the patient is returned to bed after operation, proctoclysis is instituted and maintained until the serious symptoms of intoxication cease. The continuous method is by far the most scientific and successful. Moderate distention is the normal condition of the large intestine. If it is hyperdistended, it causes spasm and expulsion of material. The mucosa of the large intestine absorbs water with great rapidity. The retention of fluid in the colon depends entirely upon the method of its administration. We have visited hospitals numbers of times, and have been shown patients who are receiving the Murphy treatment. We should not have recognized it without the label. It is difficult to impress those administering it with the importance of detail, notwithstanding that the best results are secured only by close attention to detail. A fountain syringe, to which is attached a three-eighths-inch rubber hose, fitted with a hard rubber or glass vaginal douche tip with multiple openings, was the medium originally used. The tube should be flexed almost to a right angle three inches from its tip. A straight tube must not be used, as the tip produces pressure on the posterior wall of the rectum when the patient is in the Fowler position. The tube is inserted into the rectum to the flexion angle, and secured in place by adhesive strips binding it to the side of the thigh, so that it cannot come out; the rubber tubing is passed under the sheet to the head or foot of the bed

to which the fountain is attached. It should be suspended from six to fourteen inches above the level of the buttocks, and raised or lowered to just overbalance hydrostatically the intra-abdominal pressure—*i.e.*, it must be just high enough to require from forty to sixty minutes for one and one-half pints to flow in, the usual quantity given every two hours. The flow must be controlled by gravity alone, and never by a forceps or constriction on the tube, so that when the patient endeavors to void flatus or strain, the fluid can rapidly flow back into the can; otherwise it will be discharged into the bed. It is this ease of flow to and from the bowel that insures against over-distention and expulsion on to the linen.

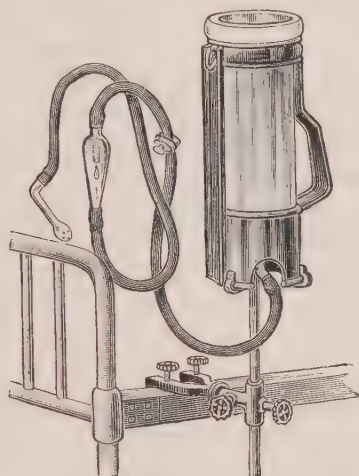


Fig. 1.—Enteroclysis apparatus, showing attachment to side of bed.

“The fountain had better be a glass or graded can, so that the flow can be estimated. The temperature of the water in the fountain can be maintained at 100° by casement in hot-water bags. The fountain is refilled every two hours with one and one-half pints or two pints of solution. The tube should not be removed from the rectum for two or three days, except for bowel movement. When the nurse complains that the solution is not being retained, it is certain that it is not being properly given; even children tolerate proctoclysis surprisingly well. We have administered as much as thirty pints of salt solution in twenty-four hours, and it was all retained. We be-



lieve that, next to the conservative technic of the operative procedure, proctoclysis is second in importance as a life-saver. It rapidly restores blood-pressure, it improves the capillary circulation, it quiets the thirst, it eliminates the septic products and increases the secretions. All the details are simple, but they must be carried out with precision to secure the best results."

*Gastric Lavage.* Having assisted nature by drawing off the infectious agents of the peritoneal cavity by means of gauze wicks and by dilution of toxic products through the use of enteroclysis, we also attempt to remove the poisonous agents through the stomach, which organ is very active in itself in throwing off foreign substances from the abdomen. Evidence of this elimination is shown by vomiting during the acute stage of the disease. The marked depression of the patient after operation or during early convalescence can be attributed to the efforts of the stomach in eliminating toxic products of the abdomen. By washing out the stomach, however, immediately after operation, the toxic products are rapidly disposed of, and physical depression is thereby avoided. Gastric lavage is an essential part of the so-called "Ochsner treatment," which aims to abate the abdominal infection by withholding all food, thereby reducing intestinal peristalsis, and by eliminating infectious products through the stomach. By washing out the stomach, reverse peristalsis is checked, vomiting ceases, and there is a general decline of all the constitutional symptoms, together with a tendency of the infection to localize itself at its seat of origin. Vomiting will continue if the stomach contains the slightest amount of intestinal products or poisons absorbed from the peritoneum. Lavage must therefore be continued at repeated intervals until the irritability of the stomach ceases.

The technic of gastric lavage may be described as follows: The patient is placed on his side and the throat sprayed with a 4 per cent. solution of cocain two or three times within a period of five minutes. The stomach-tube, after a thorough cleansing, is placed in a basin containing cracked ice or ice water, remaining so immersed until it is thoroughly cool. The tip of the tube is then placed in the mouth and gently directed toward the pharynx. The patient is requested to swallow re-

peatedly, until the esophageal muscles take a firm hold on the tube, guiding it into the stomach. That portion of the tube remaining outside is lowered in order to siphon off the stomach contents. A pint (473 mls) of warm water or saline solution is poured into the stomach, and then siphoned off. This may be done repeatedly until the siphon liquid returns clear.

Gastric lavage may be performed before and after operation, and in cases of acute gastric dilatation, which sometimes occurs after general anesthesia. It must be repeated also until the acute threatening symptoms subside. The contraindications of gastric lavage are carcinoma and ulcer of the stomach.

*Feeding and Stimulation.* Prior to operation all food and drink should be absolutely withheld. Feeding increases the burden on the stomach, tends to produce vomiting and abdominal distention, and may aggravate the constitutional symptoms. Following operation, all food is withheld until the acute distressing symptoms have subsided. Thirst is controlled, however, by enteroclysis, and the dryness of the lips may be overcome by moistening them with a solution of glycerin and lemon juice. If the heart is weak, the pulse rapid and feeble, stimulation may be called for by the administration hypodermically of strychnin sulphate,  $\frac{1}{30}$  grain (0.00216 Gm.), every three hours. Tincture strophanthus or tincture digitalis may also be given every three hours. Where rapid stimulation is desired, a hypodermic of camphor, 2 grains (0.130 Gm.) in olive oil, may be given. If the pain is severe following operative interference, the use of morphin is indicated. After the first twenty-four or thirty-six hours following operation, the general condition of the patient should be materially improved. Continued alarming symptoms after continuous enteroclysis, gastric lavage, and proper drainage through the abdominal wound, indicate an extension of the infection to the general blood-stream, resulting in blood-poisoning or septicemia. Should the patient feel much relieved, however, if the temperature has declined, and the abdominal drain is filtering the toxic products of the abdomen, as shown by moist and foul-smelling dressings, and if the nausea has ceased, feeding by mouth may be commenced. Albumin water, liquid peptonoids, skimmed milk, beef broths, and orange juice may be given in divided portions. Sometimes rectal feeding must be resorted to if the

stomach remains irritable. An appropriate rectal feeding may consist of the yolk of one egg, 1 tablespoonful (15 mls) of liquid peptonoids, 1 tablespoonful (15 mls) of whiskey, and 4 ounces (120 mls) of peptonized milk, administered twice daily during the intervals of enteroclysis.

Regarding the administration of raw eggs, recent experiments have contradicted our usual opinion on this subject. Bateman<sup>2</sup> cites numerous instances to show that raw eggs may cause diarrhea and vomiting, and that the utilization of egg white protein in the alimentary tract is often found to be as low as 50 per cent. If this be true of the normal digestive tract, it is reasonable to suppose that the stomach and intestines, already hampered by an inflammatory process, will only be able to take care of a much smaller percentage of egg protein.

*Position of Patient During Acute Attack of Peritonitis.* Mention has already been made of the posture of patients suffering from acute inflammation of the peritoneal cavity. The mesenteric and diaphragmatic portions of the peritoneum are the areas most actively engaged in absorption, while the pelvic peritoneum is least active. The patient should, therefore, be placed in such position as will cause the exudations in the peritoneal cavity to gravitate to the most dependent parts, where absorption is very slow, prohibiting the extension of the inflammatory process. Fowler has recommended a semi-recumbent position, midway between sitting up and lying down. This has become a recognized method of treatment in acute abdominal conditions, and is known as "Fowler's position." Even before operation the patient may be placed in this posture, and when moved from the ward to the operating-room. A prone position is only adopted after the acute inflammatory process has subsided.

*When to Stop Enteroclysis.* The abdomen is capable of absorbing 10 per cent. of the body-weight in thirty minutes, according to Robinson. Only sufficient normal solution is required during the process of enteroclysis to wash out the abdomen, to dilute the toxins, to allay thirst, and to activate the kidneys. Where the infection is severe, however, a large quantity of solution may be necessary. When distress follows enteroclysis, it is due either to rapid administration, to distention

of the rectum, or to high pressure from the fountain syringe preventing the regurgitation of flatus or rectal contents. When the abdominal dressings and drains become moist, without much staining and with little or no odor, when the pulse becomes full, regular and strong, and when the temperature is declining toward normal, it is safe to assume that enteroclysis has already performed its usefulness.

*When to Give Cathartics.* Constipation is frequently present during acute peritonitis. This results from a "splinting" of the bowel, which is Nature's method of preventing the spread of infection. Just prior to operation the rectum may be emptied by an enema consisting of  $\frac{1}{2}$  pint (236 mls) of salt solution. After operative interference the bowels are in a state of stasis, and peristalsis is very inactive. The generation of gases in the intestines results, and is attended with abdominal distention. The distress following the accumulation of gases is oftentimes very annoying. Relief may be obtained by the insertion of a rectal tube and by the hypodermic administration of eserín sulphate,  $\frac{1}{60}$  grain (0.00108 Gm.), every three hours. Abdominal distention may also be relieved by an asafetida enema. On the third or fourth day following operation a dose of castor oil may be given.

### CHRONIC PERITONITIS.

*General Considerations.* When the abdominal viscera is subject to mildly inflammatory disturbances continued over a long period of time, there is a gradual formation of connective tissue, which affects not only the organs themselves, but the adjacent peritoneum. In the case of the liver, the increasing fibrous tissue which is common in hepatic cirrhosis obstructs the abdominal circulation so as to affect the entire peritoneum, which becomes thickened and opaque.

An acute inflammatory process of the intestines or mesentery may subside, causing adhesions to the abdominal wall or the adjacent viscera, which are localized forms of chronic peritonitis. The peritoneal lining may also become infected with tuberculosis. This is a chronic, slow, degenerative inflammation, attended with or without exudation, causing adhesions and matting together of the abdominal viscera. Any foreign body may set up a slow inflammatory process, causing



symptoms of distress without systemic signs. A gauze sponge or a hemostat left in the abdomen following operation may produce an aseptic inflammatory condition, resulting in new fibrous formation, which causes indefinite, vague, and annoying abdominal symptoms. The use of chemical or antiseptic agents in abdominal surgery may also bring about an inflammatory process in the peritoneal cavity, resulting in the formation of adhesions between the intestines and abdominal wall.

Chronic peritonitis may be local or general, depending upon the extent and degree of the irritating cause. Localized peritonitis is usually the result of an acute localized peritonitis which has subsided. The symptoms are oftentimes vague and indefinite in character, but produce very much discomfort and misery. Distress and sensations of pulling and drawing at definitely located places in the abdomen are the prominent complaints. The patient usually wanders from one physician to another, with little or no relief from medical treatment, until radical operative measures are adopted.

The clinical pathology of a localized peritonitis is most often shown *post mortem*, at autopsy, for it presents few, if any, symptoms during life, and is seen as fibrous bands extending between coils of the intestines and as adherent strands between the abdominal viscera and the abdominal wall. In diffuse adhesive peritonitis the peritoneal cavity is practically obliterated, the intestines are matted together, and adhere closely to the mesentery. It may often be difficult to separate the parietal from the visceral peritoneum. This condition is usually seen in the dry form of tuberculous peritonitis. There is also a chronic form, in which the peritoneal lining is thickened, and presents a white, opaque, glistening surface, with more or less exudation of serum. This is usually found in hepatic cirrhosis of long duration, attended with an obstruction of the abdominal circulation. Another form of chronic inflammation is presented by a thickening of the intestinal walls and mesentery, which are intimately matted together, and roll up in a large ball, situated between the stomach and the colon.

Tuberculous peritonitis probably is the most important from the standpoint of surgical treatment. It is always secondary to tuberculosis elsewhere, as in the fallopian tubes and the retroperitoneal glands, or it is a part of an acute miliary tuber-



culosis. In cases of infection by the tubercle bacillus alone there is an exudation of serum attended with abdominal distention, the degree of which will depend upon the amount of exudation. In mixed infections, however, where other micro-organisms are present in conjunction with the tubercle bacillus, the inflammatory process usually assumes the adhesive type, which is due to the activity of the complicating organ-



Fig. 2.—Tuberculous peritonitis. Surface of liver, stomach, and intestines studded with tubercles.

isms. When the tuberculous process has its origin in the intestines, the infection nearly always is a mixed one, and the pathologic changes consist of a matting together of the intestines and mesentery. Tubercles may or may not be found upon the peritoneal surfaces.

Localized peritonitis may or may not show *symptoms* about the part affected. Digestive disorders arise from a chronic

inflammatory process in any part of the abdomen, and may be accompanied by numerous nervous symptoms bordering upon neurasthenia. The history of a previous operation or of a former acute inflammatory process may help to locate the seat of the chronic trouble. Adhesions about the appendiceal region may give rise to dull, dragging pains, attended with loss of weight, irritability, and sometimes mental depression. Very



Fig. 3.—Tuberculous peritonitis, showing extensive matting of intestines.

often pain is entirely absent, and the case remains obscure with indefinite and vague symptoms. Physical signs may be absent except in post-operative cases, where the scar may assist in locating the origin of the trouble.

In general peritonitis the symptoms come on gradually. The patient may complain of either constipation or diarrhea; there is loss of weight, anemia, and distress over the abdomen;

digestive disturbances are frequent; there is loss of appetite, aversion for certain foods arises, and general neurasthenic symptoms are prominent. Pulmonary, glandular, or bone tuberculosis may precede infection of the peritoneum.

The abdomen either is distended by serous fluid or it may be scaphoid, firm and stiff, as the result of contracted tissues beneath. Palpation of the abdominal wall may detect the matted condition of the intestines, which simulate a tumor mass. Percussion dullness depends upon the presence of fluid or the thickening of the mesentery. When fluid is present, the percussion dullness will vary with the posture of the patient. Encapsulated exudations may give rise to dull areas in various parts of the abdomen interposed by tympany of the distended intestines. Gradual distention of the abdomen, which is nearly always caused by tuberculosis, is attended with anemia, loss of weight, and sometimes with an evening temperature. It occurs most frequently in the female, and points to pelvic origin.

#### TREATMENT.

The prevention of adhesions should be given first consideration in the treatment of local peritonitis. Walker<sup>3</sup> and Ferguson have described the formation of adhesions as due to the production of fibrin, which is the foundation for fibrous tissue.

Adhesions could be prevented, according to their advanced theory, if coagulation of the exudate resulting in the formation of a fibrinous mass is avoided. Fibrin is formed by the action of thrombin on fibrinogen, which ordinarily takes place in shed blood. There are two factors necessary for this action to take place: first, that blood in the absence of its calcium content remains fluid; and, second, that there is some undetermined substance in the nature of a kinase which reactivates the inactive prothrombin of the circulating blood to form the active thrombin. It is known as fibrin formation in shed blood and can be delayed indefinitely by the addition of citrates or oxalates to hold the calcium. The authors of this theory have concluded that hypotonic salt solutions do under certain conditions prevent peritoneal adhesion after laparotomy, and that the best solution is composed of sodium citrate 3 per cent. and sodium chlorid 1 per cent. for clean laparotomies. It is recommended that 500 to 600 mils (16.9 to 20.2 f $\bar{5}$ ) of this solution

be introduced into the abdominal cavity. When packing off the intestines, it is advised to wet the gauze with this citrate solution. This theory, however, must be thoroughly tried. It is mentioned here because of its acknowledged importance.

The operator must take ample precautions to prevent traumatism of the peritoneum and the serous coat of the intestines or other viscera, otherwise the resulting adhesions may offset the advantages of the local operation. An extra amount of lymph is thrown out by the abused tissues, which adhere to the adjacent structures for protection. When the stomach, intestines or omentum are lifted out of the abdomen for any purpose during operative procedures, they should be protected from drying by the application of hot moist compresses or by thin sheets of rubber dam. Dry gauze is irritating to these structures and stimulates connective tissue formation, with resulting adhesions. Rough handling and frequent sponging of the peritoneum also injure this delicate structure, and predispose to adhesion formations.

#### **TREATMENT OF LOCAL CHRONIC PERITONITIS (ADHESIONS).**

A large proportion of those suffering from chronic constipation are so affected because of peritoneal adhesions. In a great many autopsies, regardless of the cause of death, there are invariably found isolated adhesions in the abdomen which have been unsuspected during life and have produced few symptoms or none at all. When a patient complains of vague and indefinite symptoms in a localized area in the abdomen, there is strong suspicion of the presence of adhesions. When these symptoms become distressing to the patient, there is but one measure of relief and that is surgical interference. This consists of breaking up the adhesions by stripping them from the affected viscera. This, however, leaves new raw surfaces, which may form adhesions again if not properly protected in the course of operation. Morris<sup>4</sup> prevents the formation of new adhesions by an aristol film or by the use of a Cargile membrane made from the sterilized peritoneum of the ox. The aristol film is formed by sprinkling aristol powder over the denuded surfaces from which the adhesions have been separated. The lymph is thereby incorporated with the aristol



and forms a protective coating for the raw surfaces. The Cargile membrane is placed over the denuded surface, and by natural cohesion is approximated to the raw areas. If this tissue does not adhere, it may be held in place by fine catgut. Absorption of this membrane takes place after the surfaces exposed by the detachment of adhesions are healed. Sterile oil has also been advocated to prevent the formation of new adhesions. Regardless of which method is used, there is always danger of new adhesions forming. Therefore, the surgeon should use as little force as possible in removing old adhesions in order to reduce the possibility of new tissue formation.

#### TREATMENT OF CHRONIC GENERAL PERITONITIS WITH EXUDATION.

Chronic peritonitis with exudation of serum is nearly always a tuberculous process. The treatment is surgical, and consists of opening the abdomen, removing the source of infection, which may be the fallopian tubes, the appendix or enlarged abdominal glands, and mopping out the excess fluid in the peritoneal cavity. Frequently no local lesion is found, and a mere laparotomy together with the removal of fluid is sufficient to cause a complete change in the pathologic condition of the peritoneum, resulting in a more or less permanent cure. About 40 to 50 per cent. of cases of serous peritonitis are cured by simple laparotomy.

Some surgeons irrigate the abdominal cavity with salt solution, others dust the peritoneum with iodoform, while still others make use of iodine solutions, or inject oxygen on the assumption that this element has a beneficial effect on the tuberculous process. It is usually taught that there is some exciting factor in the air which, when admitted to the abdominal cavity, brings about a complete cure. Laplace explains this on the ground that a severe impression is made upon the entire body by the operative interference, as is sometimes the case in epilepsy, where surgical means may bring about a profound change in the individual, attended with more or less improvement of the epileptic condition. Mayo<sup>5</sup> claims that the cure resulting from simple laparotomy is due to the fact that the withdrawal of fluids from the abdomen permits the fallopian tubes to come in contact with some neighboring part of



the peritoneum, where they become adherent and closed, thereby ceasing to drain tuberculous products into the peritoneal cavity. These occluded tubes may slowly develop into abscesses, which, predisposing to a general tuberculosis, may be a source of danger. In all cases of tuberculous infection of the peritoneum, whether the local lesion is in the tubes, intestines, appendix, or abdominal glands, operation is always performed without drainage; otherwise secondary infection may take place with resulting fistulæ and septic infection. This principle also holds true of the surgical treatment of tuberculosis elsewhere. The older writers call attention to the fact that spontaneous evacuation of abscess of tuberculous origin gives better results than incision with drainage, which invariably leads to a mixed infection and a chronic unyielding process.

### NON-EXUDATIVE CHRONIC GENERAL PERITONITIS.

In cases where the symptoms of digestive disturbances attended with colicky pain and chronic constipation are persistent and annoying, it is sometimes necessary to resort to operative interference to remove the exciting cause. On opening the abdomen, inspection is made of the various viscera, and if adhesions are found, these must be removed by stripping or by resection. A constricted bowel should be released from its adherent structures or removed by complete resection of the affected parts. Lane recommends the removal of a large portion of the bowel in suitable cases. Preceding the operative interference, however, the patient may be assisted by the administration of various liquid paraffin preparations. These lubricate the bowel, soften the stools, and tend to relieve many of the distressing symptoms. Where there is exudation, the internal administration of cathartics and diuretics may be of value in reducing the peritoneal fluid. Tuberculin may also be used in increasing doses, depending upon the reactions presented by the patient in cases of tubercular infection. Medical measures should also be used after operative interference, when general hygienic measures should be advocated. These patients require out-door exercise, restricted and carefully regulated habits, and plenty of fresh, wholesome, nourishing food.

## PELVIC PERITONITIS.

Because of the intimate relation of the peritoneum to the pelvic organs, inflammatory conditions arising in the pelvic viscera may extend to the lower peritoneal cavity, producing a local pelvic peritonitis. The viscera are so surrounded and so protected by supporting ligaments that inflammatory processes are usually confined to the seat of origin. Very often the pelvic inflammation has its beginning in the uterus and extends through the fallopian tubes to the peritoneum. Infection may, however, travel by way of the lymphatics directly through the uterine walls or by way of the venous sinuses, which may carry the infectious agents into the general blood-stream. Injury or rupture of any of the pelvic viscera, caused directly or indirectly, may bring about a state of local peritonitis. In the female, however, peritoneal infection practically always has its origin in the uterus.

Inflammations of the pelvic organs which are severe enough to involve the peritoneum must necessarily be accompanied by distinct *pathologic changes*. If the tubes alone are affected, the fimbriæ are matted together, the tubes are closed and distorted, and may be adherent to adjacent structures by exudation. This exudate may be large or small, depending upon the severity and extent of the infection and the type of micro-organism prevailing. A rapidly spreading process may have little or no exudate, while a subacute infection may have an abundant serous or seropurulent exudation. If no abscess has been formed, all the pelvic organs may be matted together in an irregular mass with purulent material in the pockets of connective tissue. In other instances the cellular tissues surrounding the uterus may be the seat of a round-cell infiltration as the result of infection from the cervix. The amount and character of the exudate also vary with the type of infection, and may be classified as serous, serofibrinous, or purulent.

Infectious thrombosis of the veins following labor or premature birth may be the cause of a severe pelvic inflammation extending from the sinuses of the uterus into the veins of the broad ligaments and ovaries. Such an infection may result in a pelvic abscess, may extend by continuity of tissue to the abdominal peritoneum causing a general peritonitis, or may



Fig. 4.—Pelvic peritonitis (tuberculous). Abscess in cul-de-sac of Douglas. Bladder implicated alone.

lead to an infection of the general blood-stream producing septicemia. In the the male, rupture of the bladder or a prostatic abscess may lead to pathologic changes with exudation, depending upon the severity of the case.

Pelvic peritonitis is usually superseded by *symptoms* referable to one or more of the pelvic organs. Pain in the lower abdomen is the cardinal symptom. The patient usually takes to bed because there is difficulty in walking, sitting down, or pain on even slight movement. The temperature varies with the degree of infection, ranging from 100° to 105° (37.8° to 40.6° C.). The higher temperatures are indicative of streptococcic infection following labor or abortion, or of rupture of one of the pelvic organs.

Every case of pelvic inflammation in the female gives a history either of labor, abortion, chronic endometritis, gonorrhea, or instrumentation. Inspection of the abdomen reveals a limitation of the respiratory movements, more especially about the part affected. In severe cases the entire abdominal wall may appear stiffened, while the respiratory movements of the chest are accelerated to compensate for the impaired abdominal wall. Palpation reveals tenderness in one or both inguinal regions or over the whole abdomen; even a board-like rigidity may be found in severe cases. By percussion over the tubal areas more or less organized masses may be outlined as dull areas surrounded by tympany. Vaginal examination nearly always detects a discharge, the character of which may lead to suspicion of the type of infection. Following labor or miscarriage the usual signs of injury are detected. Tenderness over the tubes or uterus may be intensified by a vagino-abdominal palpation. An abscess mass may be outlined on either side of the abdomen behind the uterus. Such a collection of pus gives a sense of firmness to the palpating finger, is more resistant to pressure than the surrounding tissues, and is very tender to touch. Abscesses may localize in the cul-de-sac of Douglas, may be situated high in the pelvis, or may extend completely around the uterus. When the abscess is large, fluctuation is easily detected. An early forming abscess may be found by careful rectal examination.

The usual septic symptoms, such as chills, fever, and sweat, are present in pelvic peritonitis, attended with abscess

formation. In cases of septic thrombosis, pain, tenderness, and fever may be present without evidences of localized lesions.

The *diagnosis* of pelvic peritonitis must be differentiated from that of acute appendicitis, tubal pregnancy, acute endometritis, a tumor mass or cyst twisted on its pedicle, infected dermoid cysts, and necrotic fibroids.

*Appendicitis* usually gives the history of digestive disturbances, while pelvic inflammation presents symptoms referable to the uterus accompanied by metrorrhagia, dysmenorrhea, and vaginal discharge. An inflammatory process in the lower abdomen confined to the right side, occurring in girls and unmarried women, is usually indicative of appendicitis.

In *tubal pregnancy* there is usually a history of long sterility, cessation of menstruation which had previously been regular, sudden onset of pain, and persistent bloody discharge from the vagina for one or two weeks; the fever may be slight or even absent, and other signs of pregnancy are present.

Cases of pedunculated *pelvic tumors*, gangrenous as the result of twisting or torsion, may give rise to a condition resembling pelvic peritonitis. In the former, however, a history of tumor growth of gradual formation is obtained. The pain comes on suddenly with no premonitory symptoms. Severe shock takes place immediately. Later there may be fever resulting from the degenerative process in the tumor mass, caused by obstruction of its blood-supply. The absence of other clinical symptoms especially referable to the uterus and its appendages should be considered among the diagnostic points.

A suppurating *dermoid cyst* may bring about a local peritonitis. The absence of uterine infection in a woman who has never been pregnant, presenting symptoms of a growing tumor in the ovarian region, should lead to the suspicion of dermoid cyst.

A *fibroid tumor* undergoing degenerative change may also simulate a peritoneal inflammation, but is distinguished by its characteristic symptoms—mainly, menorrhagia, leucorrhea, pressure-symptoms, local pain, and a growth increasing in size in the lower abdomen.



### TREATMENT OF PELVIC PERITONITIS.

When a peritoneal inflammation is confined to the region of the affected organ, the treatment is limited to a localized seat of origin. Each case must be treated according to the type and extent of the lesion present. General measures which apply to all forms of regional pelvic peritonitis may be enumerated as follows: Rest in bed, the use of laxatives as indicated in the individual case, and hot douches to lessen the amount of vaginal discharge and inflammatory process by stimulating the blood-vessels and lymphatics. In the case of the old and feeble and in the young, heat may be applied to the part with great relief, while in the robust and in cases of violent and rapidly spreading infections cold is advisable. The choice of heat and cold rests with the individual case; whichever gives the greater comfort and relief should be used. If the temperature is high, an alcohol sponge or a warm-water sponge may bring about an appreciable decrease. The use of sedatives should be held in abeyance unless operative interference has been decided upon. Opiates or other sedatives mask the true diagnostic symptoms of the disease, and tend to mislead the surgeon in choosing the time for operation. When pelvic peritonitis follows labor or abortion, this is an indication of infection by reason of retained fragments of the after-birth or carelessness in aseptic precautions in handling the patient. The treatment of such cases should be directed toward cleaning the uterus of its infected products, either by the finger or the curet. Any accumulation of pus in the cul-de-sac of Douglas calls for evacuation by incision through the posterior vaginal wall. Drainage should be established by the introduction of a large stout drainage tube, which is held in place by packing with sterile gauze. The entire vagina is also filled with sterile gauze to collect the discharging pus. This vaginal packing is removed daily, while the drainage tube is allowed to remain until granulation has obliterated the abscess cavity. Irrigation of the abscess is not only unnecessary, but may be a dangerous procedure should the retaining walls of the abscess give way under the pressure of the irrigating fluid, and thus infect the abdominal cavity.

When the abscess mass is detected high in the pelvis, and the inflammatory process is not fulminating, it may be advisable to wait until the acute symptoms subside, provided that the physical condition of the patient is good. Pelvic conditions frequently take care of themselves during the acute stage, localizing the infection, when conditions become more favorable for operative interference. It should be remembered, however, that violent, acute, fulminant, and highly toxic inflammations are dangerous to the life of the patient, and that waiting for these symptoms to subside may be disastrous. Immediate surgical interference should, therefore, be adopted in such cases. Very often mild infectious processes of the pelvis undergo resolution by ordinary general measures, which consist of douching, curetment, rest in bed, and the use of laxatives. Following the subsidence of the early acute symptoms, surgical interference may be adopted to relieve the local infection. When the inflammatory process spreads rapidly, the peritoneal cavity must be opened immediately, either by a vaginal or abdominal section. If the former method be chosen, the posterior wall of the vagina is incised, the peritoneal cavity opened, and a drainage tube inserted and held in place by suitable packing. The drainage tube is not removed until the purulent discharge ceases and the acute inflammatory symptoms have subsided.

Abdominal section in the median line also may be performed in extensive inflammatory conditions of the pelvis. This procedure is adopted when the peritoneal infection has extended from the pelvis and invaded the abdomen. This permits the surgeon to reach pockets containing infectious material not readily drained through the vaginal incision. Where the inflammatory process is extensive, it may be advisable to make a vaginal incision in addition to the abdominal opening.

The other measures following drainage in pelvic peritonitis are similar to those already mentioned under acute general peritonitis, which consist of limiting the infectious process by discouraging peristalsis, accomplished by withholding food and drink; of the elimination of infectious material by enteroclysis; and of limiting the absorption of toxic products by resorting to Fowler's position and gastric lavage (*q.v.*).

There are many cases of pelvic peritonitis following labor

and abortion which are treated medicinally with complete recovery. Adhesions may occur about the inflammatory parts, but in spite of these complications complete recovery is often regained. This does not hold true of gonorrheal infections, which may cause chronic pelvic inflammation, sterility, and chronic invalidism. Repeated attacks of pelvic peritonitis may arise from foci of chronic infection which become active from time to time in the pelvis, attended with menstrual disturbances and pain on exertion. Surgical interference is usually indicated to prevent the recurrence of these attacks.

### APPENDICULAR PERITONITIS.

Inflammatory lesions of the appendix affecting its entire structure are practically always associated with a local peritonitis. The degree of peritoneal inflammation depends upon the severity of the appendiceal infection, the type of the infecting micro-organism, and the resistance of the patient. Being a localized condition, it is reasonable to suppose that the peritonéum in the immediate vicinity of the appendix has thrown out sufficient exudate to limit the inflammatory process. The pathologic findings, therefore, are those of a severe appendicitis together with more or less exudation of serum, fibrin or pus, depending upon the stage of the inflammation. The appendix may be either free or bound down to the adjacent tissues by exudate, it may be hidden in an abscess mass, or separated from its base as the result of a gangrenous process.

The peritoneal implication always is dependent upon an antecedent inflammation of the appendix. A repeated mild catarrhal inflammation of the appendix may lead to extension of the inflammatory process to the peritoneum. It is necessary, however, that the infection be of a severe or moderate type in order to reach the peritoneal cavity.

The *symptoms* of appendicular peritonitis are those of acute appendicitis together with those of a local peritonitis. There is pain, tenderness, and rigidity over and surrounding the appendiceal area. Fever is high, ranging from 102° to 105° F. (38.9° to 40.6° C.), and a localized lesion may be palpated through the abdominal wall. There are instances in which no localized mass can be detected, but there is rigidity, redness,

and tenderness, centered about McBurney's point, or at the intersection of a line drawn from the navel to the antero-superior spine of the ileum, with a second line vertically placed corresponding to the outer edge of the right rectus muscle.

There is a high leucocytosis ranging from twelve thousand to fifteen thousand cells to the cubic millimeter. The resistance of the patient must be great enough in appendiceal peritonitis to localize the infection. This would infer that phagocytosis has been sufficiently active to produce an increased leucocyte count. More especially is this true in cases of abscess formation. The diagnosis of peritonitis circumscribed to the appendix is made upon the severity of the localized symptoms of severe pain, tenderness, rigidity, abdominal distention, and upon the constitutional signs such as fever and more or less prostration.

The *treatment* should be directed toward the original seat of infection, the appendix. Operative interference is practically always indicated, and the earlier it is performed the better the prognosis. In the large majority of cases the appendix should be removed, but where there is danger of disseminating infection or where the tip of the appendix is sloughed away, it is better to resort to simple drainage. The abscess should be thoroughly cleaned out, and adequate drainage provided for by the use of gauze packing and drainage tubes. Other measures of elimination, such as the "Murphy enteroclysis" and gastric lavage, should be used as the individual requires. (See p. 767.) The treatment of appendiceal peritonitis by medical means is no longer advocated. Although many cases of mild appendicitis recover after the application of heat or cold, rest in bed, and the withholding of food, these are but few as compared with those which sooner or later require immediate surgical interference.

### SUBPHRENIC ABSCESS.

When one of the abdominal viscera is subject to an acute inflammatory process attended with leakage of its contents into the abdominal cavity, an abscess may form, which by process of least resistance centers itself beneath the diaphragm, and is known as a subphrenic abscess. By far the most frequent cause of such phlegmonous accumulations is ulcer of the stom-

ach, which, allowing the gastric contents to reach the peritoneal cavity, brings about a purulent infection beneath the diaphragm. A suppurating appendix or a duodenal ulcer also may be the source of origin of such an abscess. Among other causes may be mentioned inflammatory conditions of the biliary passages, spleen, pancreas, kidney, liver, vertebræ, and pleura.



Fig. 5.—Subphrenic abscess originating in the liver.

The *symptoms* vary according to the direction of the abscess formation and the source of its origin. When caused by a perforating gastric ulcer, the symptoms arise gradually, the infection being confined by adhesions previously formed from the gastric erosion. There is tenderness in the upper belly in the right or left hypochondriac region, with a gradual bulging beneath the ribs on either side. The rupture of a newly formed gastric or duodenal ulcer, with few limiting adhesions, or the



acute perforation of a purulent inflammatory process in any of the other abdominal viscera adjacent to the diaphragm, presents acute symptoms characterized by rapid rise of temperature, pain or tenderness in the upper abdomen, and nausea, and vomiting, consisting of stomach contents, bile, or blood.

Subphrenic abscess usually presents a hectic type of temperature, attended with chills, sweats, and rapid loss of weight. An accumulation of pus pushes the diaphragm upward, and thereby hinders the respiratory movements. The cardiac action is also quickened by pressure of the abscess and by stimulation of the toxic process. The inflammatory process may extend through the diaphragm into the pleural cavity, or may even rupture into the lung, attended with expectoration of pus and blood.

On palpation the lower border of the liver is found to be depressed, and far below its usual anatomic line. On percussion, hepatic dullness is found to extend upwards, and varies with the posture of the patient. Sometimes tympany is elicited over the bulging mass, which is due to direct communication with the stomach or to the generation of gas produced by the activity of the colon bacillus. The characteristic findings of subphrenic abscess, therefore, are, from below upwards, dullness over the liver, flatness over the purulent exudate, and tympany superimposed. The compressed lung gives evidences of hyperresonance and accentuated breath-sounds. Respiratory movements of the opposite lung are exaggerated to compensate for the side restricted by pressure of the subphrenic abscess.

Differential *diagnosis* must be made between this disease and *pleural empyema*. The latter condition points to an antecedent history of pulmonary disease attended with dyspnea, cough, and expectoration, while in the former the symptoms are referable to one or more of the abdominal regions.

*Pyopneumothorax* may also simulate a subphrenic abscess with gas formation. The thoracic condition is accompanied by acute symptoms limited to the area above the diaphragm. In subphrenic abscess, however, the predominant symptoms are localized below the diaphragm.

Abscesses in the upper abdomen do not necessarily remain localized. They burrow in the direction of the least resistance

and may point in areas distant from the seat of origin, and make themselves mostly conspicuous in areas such as the iliac fossa and the retroperitoneal spaces of the abdomen.

### TREATMENT.

The treatment of subphrenic abscess is surgical. A free incision is made over the most prominent part of the abscess and free drainage is established. By far the largest number of cures is obtained by free and active drainage of the abscess cavity. Lang<sup>6</sup> reports 47.9 per cent. of cures by surgical interference, and only 12.3 per cent. without operation. The abscess may be opened either through the abdomen or through the chest. If by the latter route, the pleura cavity must be protected from infection by suitable packing or by suturing the divided pleura to the sides of the bulging abscess before incision is made. After sufficient drainage, and after the pyogenic cavity has been filled by granulations, attention should be paid to those causative factors responsible for the abscess, such as ulcer of the stomach, ulcer of the duodenum, or inflamed appendix.

### PERITONEAL NEOPLASMS.

Tumors of the peritoneum may be primary or secondary. More frequently, however, the latter condition holds true, and is due to extension of the neoplastic growth from adjacent viscera or from distant sources through the lymphatics.

The *benign growths* usually have their origin in the subperitoneal tissues. Lipomata are either retroperitoneal or may develop from an epiploic appendix. These are prone to either calcareous or myxomatous degenerative changes. Fatty tumors, when they occur in the abdominal wall, may grow to a fairly large size, pushing the peritoneum forward.

Fibromata may be found on the parietal or visceral peritoneum and usually are small in size.

Cysts of the peritoneum may have their origin in tumors which have undergone mucoid or colloid degeneration. The tubes and ovaries are the most frequent site of cysts which push the peritoneum forward. Chylous cysts may also be found in the peritoneum, and appear as pedunculated, grape-like, small or large masses resembling little bladders. They



Fig. 6.—Retroperitoneal carcinomatous node in the upper abdomen close to the aorta.

are the result of obstruction of the lymphatic vessels. Among other benign tumors of the peritoneum are lymphangioma and chylangioma. *Ecchinococcus* cysts also are found on rare occasions in the peritoneum.

The *symptoms* indicative of benign peritoneal tumors are those of pressure, interfering with the intestinal movements, with the circulation, or with both. The size and location of the



Fig. 7.—Carcinomatosis. Liver, stomach, and peritoneum implicated.

new growth especially influences and characterizes the nature of the symptoms. Digestive disturbances, such as nausea, vomiting, and constipation, vary with the degree of pressure upon the intestines. Stasis of the blood supply accompanied with ascites also is dependent upon the degree of pressure and the size of the tumor mass. Many of these growths of a small size are diagnosed only after laparotomy.

The *treatment* of these growths is surgical, and calls for removal when the pressure-symptoms are severe.

Among the *malignant tumors* are endothelioma, carcinoma, and sarcoma. The first of these is primary in the omentum wall. Carcinoma is nearly always secondary to extension from the adjacent viscera or other parts of the body. The frequent sites of origin of cancer are the stomach, liver, gall-bladder, uterus, rectum, and breasts. Cancer of the peritoneum also may exist as nodular masses resembling tubercles about the abdominal aorta. They are distinguished from tuberculous tubercles by their firm and dense structure, and they do not caseate.

The entire abdomen may be affected with carcinoma, this condition being known as carcinomatous. In this disease the omentum is retracted, and may appear as a tumor mass the size of a man's hand. Here and there in the reduplications of the peritoneum on the gastrosplenic, gastrocolic omentum, and mesentery are multiple cancerous nodules. The surface of the liver may be sprinkled with smaller nodules resembling the icing on cakes. Digestive disturbances, loss of weight, and a cachectic appearance, all may lead to the suspicion of the disease. Tuberculosis may simulate this condition, but has in addition to the other symptoms a hectic temperature and the usual signs of the disease elsewhere. Sarcoma may also affect the peritoneum. A diagnosis is usually made after laparotomy or at autopsy.

The *treatment* is symptomatic, and should be aimed toward relieving the patient and making him comfortable. Removal of the extensive invasion is useless, for the neoplasm is sure to recur.

### ASCITES.

Obstruction of the normal blood supply to the peritoneum results in an accumulation of fluid in the abdomen termed ascites. This fluid is a transudation from the blood through the peritoneal lining, and is of a light yellow color, and either clear or turbid, depending upon the presence or absence of cellular content, bile, or blood. It resembles very much the serum of blood, from which it is derived. The peritoneum shows little or no change when the transudate has its origin



from a non-inflammatory condition. In cases of peritonitis the serous covering of the abdominal wall may be thickened and opaque. Tuberculous peritonitis with ascites has already been described. (See p. 946.) Mention is made of a chylous ascites, which consists of the milk-white exudate resulting from the obstruction of the thoracic duct.

The accumulation of fluid in the abdomen takes place gradually, and the *symptoms* must necessarily appear in like manner. A small amount of fluid gives rise to few or no symptoms aside from a sensation of fullness or weight in the abdomen; when the amount of fluid increases so as to cause marked distention of the abdomen, the symptoms become very prominent. The respirations are accelerated as a result of pressure against the diaphragm, and there is great discomfort from the sense of weight in the abdomen. The patient complains of constipation, nausea and disturbed digestion, and the pulse is accelerated, and the cardiac action is quickened by the pressure of the retained fluid. Pressure upon the kidneys and renal vessels causes a passive congestion resulting in the presence of albumin in the urine. The abdominal wall is uniformly rounded and prominent, its size depending, of course, upon the amount of fluid present. By changing the posture of the patient there is a change in the contour of the abdomen corresponding to the gravitation of fluid to the dependent parts. The skin is stretched and shiny, and its veins may be markedly distended. The umbilicus is pushed forward at the summit of the abdominal distention. The abdominal respirations are practically absent, while the chest movements are accentuated, as evidenced by the quickened movements.

The presence of fluid is detected by placing the palm of the left hand on one side of the abdomen while the fingers of the right tap lightly the other side. A sense of fluid fluctuation is transmitted by the tapping fingers to the left hand. On percussion, a flat sound is elicited in the flanks, with more or less tympany in the center of the abdomen, where the bowels have been floated by the underlying fluid. Posture, of course, displaces the area of dullness or flatness, while tympany is usually found at the superior surface. The diagnosis rests upon the detection of a movable area of dullness coupled with a history of obstruction of the abdominal circulation.

A general uniform enlargement of the abdomen must not be mistaken for an ovarian cyst or for a chronic peritonitis following inflammation of the abdomen, tuberculosis, or diseases of the female pelvic organs. A distended bladder is sometimes confused with localized ascites. This latter condition may be eliminated by catheterization.

#### TREATMENT.

The elimination of the fluid may be accomplished readily by tapping the abdomen or by the use of cathartics, diuretics, and diaphoretics. It is difficult in cases of obstructed circulation to remove the cause, which may be cirrhosis of the liver or cancerous growths impeding the portal circulation. In cases of syphilis of the liver, however, treatment should be directed toward relieving the systemic disease. When the ascites is due to abscess of the liver, carcinoma, cysts (hydatid), or enlarged glands these sources of obstruction should be removed by surgical interference. Pulmonary conditions, such as emphysema and cardiac disease with insufficiency of the circulation, must be treated accordingly. Bright's disease and malaria also may be causative factors of ascites, which must be treated by appropriate measures.

When the distention of the abdomen is great, the fluid must be drained off by tapping or paracentesis. This must be repeated as often as is necessary, more especially in cases of cirrhosis of the liver, where it may assist in re-establishing abdominal circulation. When a small amount of fluid is present, elimination may be accomplished by the use of hydragogue cathartics, more especially when the condition is due to cardiac or renal disease. Among the cathartics recommended are magnesium sulphate, Rochelle salt, jalap, gamboge, colocynth, and calomel. Elimination may also be practised through the kidneys by the use of diuretics such as potassium citrate, spiritus mindererus, and copaiba. Sweating may be encouraged by the use of hot drinks, the application of hot-water bottles, and plenty of warm covers.

Some surgeons have attempted to assist the obstructed circulation by diverting it to other channels. Talma advocates the suturing of the great omentum to the anterior abdominal wall, where a new circulation is established which drains off the ascites into the systemic blood-vessels.

*Method of Tapping the Abdomen for Ascites.* After shaving and scrubbing the abdomen, the patient is placed in an upright position or in the semi-recumbent posture. A point midway between the umbilicus and pubes is selected for the introduction of a trocar and canula; the skin surface is painted with iodine



Fig. 8.—Abdominal paracentesis. Canula in place, drawing off fluid. Note position of ungloved hand exerting pressure on abdomen to encourage flow of fluid into basin.

and anesthetized by the intradermic injection of cocaine. The bladder is catheterized before the abdomen is prepared for operation. An incision is made about  $\frac{1}{2}$  inch (12.7 mm.) long in the median line at the selected point through the skin and a firm thrust is made through the abdominal tissue with the

trocar and canula. The trocar is withdrawn and the canula depressed to drain off the abdominal fluids. Great care must be exercised to avoid puncturing the intestines—an accident sometimes unavoidable when the bowel is adherent to the abdominal wall. Careful study of the case by percussion should determine whether the intestines are free or adherent. Should the intestines be accidentally ruptured, the case should be treated just as any other perforating wound of the abdomen. After the fluid has been removed from the abdomen, the canula should be withdrawn and the opening closed by gauze compress and adhesive strips.

## BIBLIOGRAPHY.

1. Robinson: *The Peritoneum*, 1899, Ed. 3, 398.
2. Bateman: *Amer. Jour. Med. Sci.*, 1917, 153, 841.
3. Walker and Ferguson: *Ann. Surg.*, 1916, lxiii, 198.
4. Morris: *Sajous's Analytic Cyclopedia of Practical Medicine*, 1914, i, 34.
5. Mayo: *Collected Papers of Mayo Clinic*, 1912, vi, 691.
6. Lang: *Keen's Surgery*, 1908, iii, 869.

## INDEX.

- Abdominal abscess, 936  
aneurysm, 259, 262  
  prognosis, 263  
  symptoms, 262
- Abscess, abdominal, 936  
  dental, in cardiac affections, 215  
  gastric, 757  
  iliac, 849  
  of kidney, 627. See Kidney.  
  of liver, 905. See Liver.  
  periapical, 653  
  perigastric, 692  
  perinephritic, 632  
    etiology, 632  
    symptoms, 632  
    treatment, 632  
  pulmonary, 478  
    complicating pneumonia, 480  
    diagnosis, 480, 481  
    differentiated from gangrene,  
      485  
    etiology, 478  
    pathology, 479  
    physical signs, 482  
    symptoms, 481  
    treatment, 483  
      surgical, 483  
  subphrenic, 960  
    differential diagnosis, 962  
    etiology, 960  
    symptoms, 961  
    treatment, 963
- Absence of kidney, 552
- Achylia gastrica, 762
- Acid, gastritis, 762  
  hippuric, 551
- Acromegaly, 151  
  pathology, 151  
  symptoms, 152  
  treatment, 154
- Actinomycosis, pulmonary, 493  
  diagnosis, 493  
  etiology, 493  
  pathology, 493  
  treatment, 494
- Acute appendicitis, 850  
  bronchitis, 326  
  catarrhal gastritis, 750  
  cholangitis, 890  
  cholecystitis, 890  
  colitis, 845  
  dilatation of stomach, 818. See  
    Gastrectasis.
- Acute enteritis, 837  
  esophagitis, 674  
  fibrinous pleuritis, 502. See Pleu-  
    ritis.  
  gastrectasis, 818  
  gastritis, 750. See Gastritis, 757.  
  general peritonitis, 927. See  
    Peritonitis.  
  glossitis, 668  
  infectious gastritis, 755. See  
    Gastritis.  
  leukemia, 30  
  miliary phthisis, 400  
  pancreatitis, 916, 917  
  parenchymatous nephritis, 578.  
    See Nephritis.  
  peritonitis, 927. See Peritonitis.  
  phlegmonous gastritis, 757. See  
    Gastritis.  
  pulmonary tuberculosis, general,  
    400  
  simple gastritis, 750. See Gas-  
    tritis.  
  suffocative pulmonary edema, 476  
  suppurative gastritis, 757  
  thyroiditis, 107, 108  
  yellow atrophy of liver, 908. See  
    Liver.
- Addisonian anemia, 18, 19, 20, 23
- Addison's disease, 80
- Adenitis, tuberculous, differentiated  
  from Hodgkin's disease, 45
- Adenoma of adrenals, 91  
  of kidney, 639  
  of liver, 913
- Adhesions, pericardial, 222, 225  
  peritoneal, 945, 946  
    prevention, 949, 950  
    symptoms, 947  
    treatment, 949, 950
- Adrenal extract in heart block, 219
- glands, 67  
  adenoma of, 91  
  angioma of, 91  
  antitoxic function of, 70  
  benign tumors of, 91  
    symptoms, 91  
    treatment, 92  
  connections with other glands  
    and organs, 71, 72  
  chromaffin tumors of, 91  
  chromaffin substance in, 67  
  echinococcic cyst of, 91  
    (971)



- Adrenal glands, effect of removal, 69  
 fibroma of, 91  
 function, 69  
 ganglioneuroma of, 91  
 histology, 67  
 hypernephroma of, 90  
 lipoma of, 91  
 malignant tumors of, 90  
 symptoms, 90  
 treatment, 91  
 paragangliomas of, 91  
 transplantation of, 81, 82  
 tumors, 90  
 hematoma, 87  
 treatment, 88  
 hemorrhage, 85  
 hemorrhagic pseudo-cyst, 87  
 insufficiency, 72. See Hypoadrenia.  
 overactivity, 84  
 etiology, 84  
 treatment, 86  
 rests, 68, 88  
 secretion, effects on blood-pressure, 70  
 physiologic action of, 70  
 Albumin in urine, tests for, 570  
 quantitative, 571  
 reaction in sputum, 379  
 Albuminometer, Esbach's, 571  
 Albuminuria, 569  
 Bence-Jones, 570  
 etiology, 569  
 febrile, 569  
 significance of, 569  
 functional, 570  
 significance of, 569  
 tests for, 570  
 quantitative, 571  
 Albumosuria, 570  
 test for, 570  
 Alcohol in heart diseases, 285  
 in pulmonary tuberculosis, 429, 430  
 Alcoholic gastritis, 779  
 treatment, 779  
 Allen treatment for diabetes, 470  
 Alpha iodine compound, 95, 97  
 Alternation of heart, 219  
 diagnosis, 220  
 etiology, 219  
 prognosis, 220  
 treatment, 221  
 Ammonia as a cardiac drug, 286  
 Ammonium chloride in phthisis, 449  
 Amyl nitrite, 283  
 Amyloid casts, 574  
 degeneration of kidney, 618. See Kidney.  
 disease of liver, 911  
 Anacid gastritis, 762  
 Anacid gastritis, treatment, 778, 779  
 Anaphylaxis and asthma, 352, 354  
 and hay fever, 359  
 Anemia, Addisonian, 18, 19, 20, 23  
 aplastic, 20  
 classification of, 4, 5  
 due to intestinal parasites, 6  
 to gastric cancer, treatment of, 743  
 to pulmonary tuberculosis, 382, 459  
 general indications for treatment, 3  
 in chronic nephritis, 592  
 lightning pains in, 29  
 of kidney, 574  
 etiology, 574  
 treatment, 575  
 pernicious, 17. See Pernicious Anemia.  
 primary, 4  
 splenic, 47. See Splenic Anemia.  
 symptomatic secondary, 4  
 due to intestinal parasites, 6  
 etiology, 5  
 pathology, 5  
 symptoms, 6  
 treatment, 6  
 dietetic, 6  
 hygienic, 7  
 therapeutic, 7  
 Aneurysm, 259  
 abdominal, 259, 262  
 prognosis, 263  
 symptoms, 262  
 etiology, 259  
 neosalvarsan in, 264  
 pathology, 259  
 rest in, 264  
 salvarsan in, 263  
 stricture of esophagus from, 682  
 symptoms, 261  
 thoracic, 259, 261  
 cardiac dilatation in, 262  
 cardiac hypertrophy in, 262  
 symptoms, 261  
 treatment, 263  
 of pain, 265  
 Tufnel's, 265  
 venesection in, 265  
 wiring of, 266  
 Angina, Ludwig's, 674  
 etiology, 674  
 symptoms, 674  
 treatment, 674  
 pectoris, 255  
 etiology, 255, 256  
 physical signs, 257  
 prognosis, 257  
 symptoms, 255, 256, 257

- Angina pectoris, treatment, 257  
 Vincent's, 659. See Stomatitis, Ulcerative.
- Angioma of adrenals, 91  
 of liver, 913
- Anomalies of the kidney, 552
- Anorexia in pulmonary tuberculosis, 442
- Anthracosis, 486
- Antiformin method of demonstrating tubercle bacilli, 378
- Antitoxin, diphtheria, in asthmatics, 354
- Anuria, 561  
 differentiated from retention, 562  
 etiology, 561  
 symptoms, 562  
 treatment, 562
- Aortic regurgitation, 253  
 etiology, 253  
 physical signs, 253  
 prognosis, 248  
 sudden death in, 248  
 stenosis, 254  
 symptoms, 254
- Aphthæ, Bedner's, 658  
 treatment, 658
- Apthous stomatitis, 656. See Stomatitis.
- Aplastic anemia, 20
- Appendicitis, 849  
 acute, 850  
 chronic, 850  
 differentiated from pelvic peritonitis, 956  
 etiology, 849  
 indications for operation in, 851  
 peritonitis in, 959  
 symptoms, 850  
 treatment, 851
- Appendicular peritonitis, 959  
 pathology, 959  
 symptoms, 959  
 treatment, 960
- Arrhythmias, classification of cardiac, 206  
 sinus, 207
- Arsenic in anemia, 7  
 in chlorosis, 12  
 in Hodgkin's disease, 47  
 in infantile splenic anemia, 51  
 in leukemia, 37  
 in pernicious anemia, 21, 22, 23  
 in purpura, 55  
 in tuberculosis, 459
- Arteriosclerosis, 267  
 and nephritis, 268, 600  
 etiology, 267  
 hypertrophy of heart in, 268  
 pathologic histology, 268
- Arteriosclerosis, prognosis, 271  
 symptoms, 268  
 treatment, 271
- Arteriosclerotic kidney, 599
- Artery, pipe-stem, 271
- Arthritic purpura, 52
- Artificial pneumothorax in bronchiectasis, 348  
 in pulmonary tuberculosis, 423, 424, 425, 427  
 for hemoptysis, 455
- Ascites, 966  
 diagnosis, 967  
 symptoms, 967  
 treatment, 968
- Aspergillosis, pulmonary, 499
- Asthma, bronchial, 349. See Bronchial Asthma.  
 cardiac, 476  
 hay-, 351, 358. See Hay-fever.  
 thymic, 140, 142  
 treatment, 143
- Atony, intestinal, 867  
 treatment, 869
- Atoxyl, 38
- Atrophic cirrhosis of liver, 898.  
 See Liver.  
 emphysema, 362  
 gastritis, 761
- Atrophy of liver, acute yellow, 908.  
 See Liver.
- Atropin as a cardiac drug, 284  
 in gastric ulcer, 700
- Auricular contractions, premature, 208  
 fibrillation, 212  
 etiology, 212  
 in arteriosclerosis, 271  
 treatment, 273  
 prognosis, 214  
 symptoms, 212  
 treatment, 214  
 flutter, 211  
 prognosis, 212  
 symptoms, 211  
 treatment, 212
- Auriculoventricular node, 203
- Auscultatory method of determining blood-pressure, 275
- Autogenous vaccines in endocarditis, 241
- Bacelli's sign, 514
- Bacillary emulsion of Koch, 431
- Bacilli, colon, as cause of peritonitis, 933  
 tubercle, 378  
 inoculation test for, 379  
 staining of, 378
- Backwardness in children, 138

- Backwardness in children, frequency, 138  
treatment, 139  
with pineal extract, 172
- Bacterial toxins in leukemia, 40
- Bacterins in treating diseases of lungs, bronchi and pleura, 534
- Bacteriology of pleural fluids, 502
- Balneotherapy, 286, 287  
of chronic gastritis, 770
- Banti's disease, 48
- Basedow's disease, 109
- Bathing in pulmonary tuberculosis, 422, 423, 456, 457
- Baths, electric light, 587  
hot air, 587  
Nauheim, 286, 287
- B. E., 431
- Bed, Klondike, 410
- Bednar's aphthæ, 658  
treatment, 658
- Belladonna in gastric ulcer, 700
- Bence-Jones proteinuria, 570
- Benzol in leukemia, 35
- Bertini, columns of, 544
- Biliary cirrhosis of liver, 898, 899.  
See Liver.
- Biliousness, 875  
etiology, 875  
symptoms, 875  
treatment, 876  
prophylactic, 876
- Binet-Simon test, 139
- Bismuth in gastric ulcer, 700
- Blaud tabloid, 236
- Blastomycosis, pulmonary, 499
- Bleeders, 56
- Bleeding time, 53
- Blistering in pulmonary tuberculosis, 461
- Blisters, fever, 649
- Block, heart-, 216, 243  
etiology, 216  
prognosis, 218  
symptoms, 217  
treatment, 218
- Blood, creatinin in, 610  
defibrinated, injections of, 24  
diseases of, 3  
classification, 4, 5  
test, Heller's, 567  
transfusion in pernicious anemia, 23  
urea in, 608  
uric acid in, 606
- Blood-pressure, 273  
decreased, 277  
factors maintaining, 275
- Blood-pressure, importance of comparative readings, 276  
increased, 276, 277  
treatment, 277
- instruments, 274
- in nephritis, 615
- in tuberculosis, 382  
method of determining, 274  
auscultatory, 275  
normal, 270, 276
- Bougie, esophageal, 683  
technic of passing, 683
- Bovine tuberculosis, 402, 403
- Bowman, capsule of, 544
- Breath, foul, 650
- Breathing, bronchial, 396  
bronchovesicular, 396  
cog-wheel, 395  
exercises in pleuritis, 509  
contraindications, 509  
feeble, 395  
granular, 395  
in tuberculosis, 395, 396  
tubular, 396
- Breath sounds in tuberculosis, 394, 395
- Bright's disease, 578. See Nephritis.
- Bronchi, bacterin treatment of diseases of, 534
- Bronchial asthma, 349  
and anaphylaxis, 352, 354  
and emphysema, 362, 367  
diphtheria antitoxin in, 354  
etiology, 349, 353  
pathology, 349  
symptoms, 350  
tests for protein sensitization in, 352  
cutaneous, 352  
intracutaneous, 353  
treatment, 351  
between attacks, 355  
by defibrinated blood, 355  
by desensitization, 354  
of attack, 356  
vaccines in, 355
- Bronchial breathing in tuberculosis, 396  
casts, 337, 338, 339
- Bronchiectasis, 339, 473  
etiology, 340  
hemorrhages in, 342  
pathology, 340  
physical signs, 342  
symptoms, 341  
treatment, 343  
by artificial pneumothorax, 348  
by inhalations, 344  
by intratracheal injections, 345

- Bronchiectasis, treatment, climatic, 349  
     postural, 346  
     vaccine, 345  
   X-ray diagnosis of, 343  
 Bronchitis, acute and subacute, 326  
     etiology, 327  
     in tuberculosis, 447  
     pathology, 326  
     prophylaxis, 328  
     symptoms, 327  
     treatment, 328  
       abortive, 328  
       drugs in, 330  
       hygienic, 330  
       inhalations in, 330  
       local applications in, 329  
 chronic, 332  
     and emphysema, 362, 367  
     etiology, 333  
     in tuberculosis, 447  
     pathology, 333  
     treatment, 333  
       climatic, 335  
       drugs in, 335  
       general, 334  
       hygienic, 334  
       of associated primary condition, 334  
       tonic, 335  
       vaccine, 336  
 fibrinous, 337  
     etiology, 337  
     pathology, 337  
     symptoms, 338  
     treatment, 339  
   in pulmonary tuberculosis, 447  
 Bronchovesicular breathing in tuberculosis, 396  
 Bubbling râles in tuberculosis, 397  
 Bundle of His, 203  
  
 Cachectic purpura, 52  
 Caffein as a heart drug, 285  
 Calcification of tubercles, 371, 372  
 Calcium oxalate stones, 619  
 Calculi, pancreatic, 919  
     salivary, 673  
 Cammidge reaction, 915  
 Camphor, 286  
 Cancer, green, 43  
     See also Carcinoma.  
 Cancrum oris, 661. See Stomatitis, Gangrenous.  
 Cane sugar in myocarditis, 234  
 Capsule of Bowman, 544  
 Carcinoma, frequency of, 724  
     lymphatic and Hodgkin's disease, 45  
     of esophagus, 677. See Esophagus.  
 Carcinoma of intestines, 869, 870  
     of kidney, 639  
     of liver, 913  
     of lungs, 496  
     of pancreas, 921  
     of peritoneum, 966  
     of pleura, 526  
     of tongue, 671  
 Cardiac arrhythmias, classification of, 206  
     asthma, 476  
     compensation, treatment of failing, in nephritis, 614  
     cycle, 204  
     drugs, use of, 278  
     failure, causes of, 245  
     irregularities, 202  
     physiology, 203  
 Cardiograms, 205  
 Cardiospasm, 810  
     diagnosis, 814  
     esophagitis complicating, 812, 816  
       treatment, 816  
     etiology, 810  
     forms of, 812  
     prognosis, 816  
     symptoms, 812  
     treatment, 816  
 Cardiovascular symptoms of pulmonary tuberculosis, 382  
 Cargile membrane, 950, 951  
 "Carriers" of disease, 645, 663  
     treatment, 664  
 Cathartics in acute peritonitis, use of, 945  
 Caseation in tuberculosis, 371, 372  
 Casts, 572  
     amyloid, 574  
       significance of, 574  
     bronchial, 337, 338, 339  
     epithelial, 573  
       significance of, 573  
     fatty, 573  
       significance of, 573  
     granular, 573  
       significance of, 573  
     hyaline, 573  
       significance of, 572, 573  
     leucocytic, 573  
       significance of, 573  
     significance of, 572  
     waxy, 574  
       significance of, 575  
 Catarrh of stomach, 759  
 Catarrhal enteritis, chronic, 839  
     gastritis, acute, 750  
     stomatitis, 655, 656  
 Cavity formation in pulmonary tuberculosis, 372, 397  
 Chalicosis, 486

- Charcot-Leyden crystals, 351
- Chest pains in tuberculosis, 383  
   percussion of, technic, 390  
   phthisical, 384
- Chloroma, 43  
   pathology, 43  
   symptoms, 43  
   treatment, 43
- Chlorosarcoma, 43
- Chlorosis, 8  
   etiology, 8  
   florida, 9  
   hydrotherapy in, 13  
   incidence of, 9  
   massage in, 13  
   pathology, 8  
   rubra, 9  
   symptoms, 9  
     cardiovascular, 17  
     gastro-intestinal, 15  
     nervous, 16  
   treatment, 10  
     dietetic, 14  
     of special symptoms, 15, 16, 17
- Cholangitis, acute, 890. See Cholecystitis.
- chronic, 894  
     etiology, 894  
     indications for operation, 898  
     symptoms, 894, 895  
     treatment, 895
- Cholecystitis, acute, 890  
   etiology, 890  
   limitations of medical treatment in, 898  
   symptoms, 890  
   treatment, 891
- Cholelithiasis, 892  
   chronic, 894  
     indications for operation in, 897  
     symptoms, 894, 895  
     treatment, 895  
   diagnosis, 893  
   etiology, 892  
   limitations of medical treatment in, 898  
   symptoms, 892  
   treatment, 893
- Cholera infantum, 844  
   prognosis, 844  
   symptoms, 844  
   treatment, 844
- Chorea in endocarditis, 241
- Chromaffin substance, 67, 68  
   tumors of adrenals, 91
- Chromophiles, 145
- Chromophobes, 145, 152
- Chronic bronchitis, 332. See Bronchitis.
- endocarditis, 237
- Chronic gastritis, 759. See Gastritis.
- leukemia, 30
- Chvostek's sign, 132
- Chylothorax, 529  
   diagnosis, 529  
   treatment, 529
- Chyluria, 568  
   diagnosis, 568  
   etiology, 568  
   treatment, 568
- Chronic appendicitis, 850  
   cholangitis, 894  
   cholelithiasis, 894  
   colitis, 845. See Colitis.
- endocarditis, 237  
   enteritis, 839  
   gastritis, 759  
   glossitis, 669  
   myocarditis, 230. See Myocarditis.
- pancreatitis, 918  
   peritonitis, 945. See Peritonitis.
- progressive hypoadrenia, 80  
     etiology, 80  
     pathology, 80  
     symptoms, 80, 81  
     treatment, 81
- thyroiditis, 108, 109
- ulcerative pulmonary tuberculosis, 400, 401
- Circulatory disturbances of kidney, 574
- Cirrhosis of liver, 898. See Liver.
- Citrate of iron in anemia, 12
- Climatic treatment of tuberculosis, 408
- Clubbed fingers in tuberculosis, 384
- Coagulation time, 57
- Coefficient of Haeser, 547
- Cog-wheel breathing in tuberculosis, 395
- Coin test, 532
- Cold bath in tuberculosis, 422  
   sores, 649  
   sponges in tuberculosis, 422, 423, 457
- Colic, gall stone, 893  
   renal, treatment of, 622
- Colitis, acute, 845  
   symptoms, 845  
   treatment, 845
- chronic mucous, 845  
     etiology, 846  
     pathology, 846  
     sex incidence, 845  
     symptoms, 846  
     treatment, 847
- ulcerative, 848  
     symptoms, 848  
     treatment, 848



- Colitis, ulcerative varieties, 848  
 Colloid goiter, 123  
   material of thyroid, 92, 93, 97  
 Colon bacilli as cause of peritonitis, 933  
 Columns of Bertini, 544  
 Commissures, rhagades of, 648  
 Compensation, treatment of failing  
   cardiac, in nephritis, 614  
 Compensatory emphysema, 362, 369  
   pathology of, 369  
 Complement fixation test in tuberculosis, 375  
 Congenital cystic degeneration of  
   kidney, 639  
   goiter, 120, 126  
     treatment, 126  
   pyloric stenosis, 804. See Pyloric Stenosis.  
   syphilis of liver, 912  
   valvular defects, 249  
 Congestion, pulmonary, 474  
   etiology, 474  
   pathology, 475  
   physical signs, 475  
   symptoms, 475  
   treatment, 476  
 Constipation, 854  
   as a functional neurosis, 868  
     treatment, 869  
   causes of, 854  
   diet in, 855  
   due to diminished intestinal sensibility, 867  
     treatment, 869  
   due to gastric cancer, treatment of, 745  
   in tuberculosis, 444  
     treatment, 446  
   treatment, 855  
     mechanical, 858  
     medical, 857  
 Constrictions, normal esophageal, 684  
 Constrictive goiter, 123  
 Contractions, premature auricular, 208  
   premature, of heart, 208  
     etiology, 208  
     treatment, 208  
 Corpus luteum, function of, 173  
 Cough, emetic, 378  
   in tuberculosis, 377, 446  
     treatment, 446  
 Coupling, digitalis, 214, 282  
 Creatinin, 551  
   amount, 551  
   in blood, estimation of, 610  
   origin of, 551  
   tests for, 551  
 Creosote in pulmonary tuberculosis, 448  
 Cretinism, 103  
   endemic type, 104  
   sporadic type, 104  
   symptoms, 103, 104  
   thyroid grafting in, 105  
   treatment, 104  
   Wenzel's classification of, 104  
 Cretinoids, 104  
 Crises, gastric, 826. See Gastric Crises.  
   visceral, 826  
 Crisis, Dietl's, 556  
 Croupous gastritis, 756  
 Crystals, Charcot-Leyden, 351  
 Cupping, technic of dry, 589  
 Cups, sputum, 405  
 Curshmann's spirals, 351  
 Curved finger nails in pulmonary tuberculosis, 384  
 Cycle, cardiac, 204  
 Cylindroids, 574  
   significance of, 574  
 Cystic degeneration of kidney, congenital, 639  
   goiter, 123  
 Cysts of liver, 913  
   of pancreas, 920  
   of peritoneum, 963  
 Cytology of pleural effusions, 501  
 Danger zone in tuberculosis, 397  
   zones of peritoneum, 929  
 Death, thymic, 140, 142  
 Defibrinated blood, 24, 40  
   in asthma, 355  
 Degeneration of kidney, congenital cystic, 639  
 Dental abscesses in heart disturbances, 215  
 Desensitization in asthma, 354  
 Determination of degree of activity of tuberculosis, 401  
   of degree of improvement, 401  
 Diabetes, Allen treatment for, 470  
   and hyperpituitaria, 150, 162  
   complicating tuberculosis, 470  
 Diarrhea, and enteritis, 841  
   febrile, 841  
   inflammatory, 841  
   in pulmonary tuberculosis, 445  
   nervous, 868  
     treatment, 869  
   summer, 841  
 Diet, in acute parenchymatous nephritis, 584  
   in peritonitis, 943  
   in carcinoma of stomach, 740  
   in catarrhal jaundice, 879

- Diet in chlorosis, 14  
 in chronic gastritis, 764  
 in chronic interstitial nephritis, 612  
 in chronic parenchymatous nephritis, 594  
 in cirrhosis of liver, 903  
 in constipation, 855  
 in erythemia, 61  
 in leukemia, 33  
 in tuberculosis, 416, 417  
   in children, 422  
   of kidney, 638  
 in ulcer of stomach and duodenum, 703  
 Karrell, 234  
 Lenhart, 703  
 nephritic test, 605  
 Smithies', 716  
 von Leube, 703  
 Dietl's crisis, 586  
 Diffuse vesicular emphysema, 361, 362  
   etiology, 362  
   pathology, 363  
   symptoms, 364  
   treatment, 367  
 Digestive system, diseases of, 643  
 Digitalis, 278  
   contraindications for use of, 282  
   coupling, 214, 282  
   derivatives, 279  
   dosage of, 281  
   indications for use of, 282  
   in dropsy, 281  
   physiologic action of, 280  
 Dilatation of heart, 244, 246  
   cause, 245  
   in aneurysm, 262  
   of stomach, 818. See Gastrectasis.  
 Diminished intestinal sensibility, 867  
   treatment, 869  
 Diphtheria antitoxin in asthmatics, 354  
 Diphtheritic gastritis, 756  
 Disease, Addison's, 80  
   Banti's, 47  
   Basedow's, 109  
   Bright's, 578. See Nephritis.  
   Gaucher's, 48, 49  
   Glénard's, 862  
   Grave's, 109  
   Hodgkin's, 44. See Hodgkin's.  
   Launois's, 151  
   Marie's, 151  
   Parry's, 109  
   Schönlein's, 54  
   valvular, of heart, 242. See Valvular Diseases.  
   Woillez's, 474  
 Diseases of blood, 3  
   of esophagus, 674  
   of intestines, 837. See Intestines.  
   of kidneys, 541. See Kidneys.  
   of liver, 874. See Liver.  
   of mouth, 643. See Mouth.  
   as related to systemic diseases, 643  
   of pancreas, 914  
   of parathyroids, 132  
   of pericardium, 222  
   of peritoneum, 927  
   of pineal gland, 168  
   of pituitary gland, 145  
   of salivary glands, 672  
   of stomach, 684  
   of thymus gland, 133  
   of thyroid gland, 92  
   of tongue, 668-672  
 Dispensary treatment of tuberculosis, 433, 434, 441  
 Displacements of kidney, 553. See also Kidney, Movable.  
 Dissemination of tuberculosis, methods of, 404  
 Distomatosis, pulmonary, 499  
 Diuretics in nephritis, 597  
 Diverticulum of esophagus, 679  
   diagnosis, 680  
   etiology, 679  
   prognosis, 680, 681  
   symptoms, 680  
   treatment, 680  
 Drainage in peritonitis, indications for, 939  
   technic, 939  
 Drop method of enteroclysis, 583, 940  
 Dropsy, digitalis in, 281  
   in leukemia, 42  
 Dry cupping, technic, 589  
   mouth, 672  
   pericarditis, 222, 223, 224  
 Ductless glands, diseases of, 67  
 Duodenal feeding, 714  
   in carcinoma of stomach, 742  
 Duodenum, diseases of, 684  
   ulcer of, 684. See Ulcer.  
 Dyspituitarism, 150  
 Dyspnea, toxic, in nephritis, 603  
   treatment, 616  
 Dystrophia adiposogenitalis, 160  
 Echinococcic cyst of adrenals, 91  
 Echinococcus disease of lungs, 497  
   etiology, 497  
   pathology, 498  
   symptoms, 498  
   treatment, 499

- Edema in nephritis, 581, 585, 592  
 pulmonary, 476  
   acute suffocative, 476  
     etiology, 476  
     symptoms, 477  
   etiology, 476  
   pathology, 477  
   symptoms, 477  
   treatment, 478
- Edestin test, 733
- Effusion, pericardial, 222, 225  
 pleural, 499, 509, 510. See Pleuritis, Serofibrinous, and Pleural Fluids.
- Eggs and milk in pulmonary tuberculosis, 417, 418, 419
- Egophony, 513
- Elastic fibers in sputum, 379
- Electric light bath, 587
- Electrical treatment of chronic gastritis, 773
- Electrocardiograph, 203, 206
- Emetic cough, 378
- Emetin hydrochlorid in hepatic abscess, 908  
 in pyorrhea, 654
- Emphysema, 361  
 atrophic, 362  
 compensatory, 362, 369  
   pathology, 369  
 diffuse vesicular, 361, 362  
   etiology, 362  
   pathology, 363  
   symptoms, 364  
   treatment, 367  
 hypertrophic, 362  
 interlobular, 362  
 interstitial, 362  
 large-lunged, 362  
 local, 362  
 senile, 362, 369  
   pathology, 369  
   symptoms, 370  
   treatment, 370  
 small-lunged, 362  
 types, 361
- Empyema, 521  
 complications, 523  
 diagnosis, 524, 525  
   from subphrenic abscess, 962  
 etiology, 521  
 loculated, 522  
   diagnosis, 525  
 pathology, 521  
 physical signs, 522  
 results of, 523  
 symptoms, 522  
 treatment, 524  
   operative, 526
- Emulsion, Koch's bacillary, 431
- Endameba buccalis, 651
- Endemic cretinism, 104
- Endocarditis, 236, 247  
 chorea in, 241  
 chronic, 237  
 diagnosis, 238  
 etiology, 236, 247  
 malignant, 237  
 pathology, 237, 247  
 prognosis, 239  
 treatment, 239  
   by eliminating underlying cause, 240  
   by elimination, 240  
   by rest, 239  
   during convalescence, 240  
   symptomatic, 240  
   vaccine, 241
- Endomyces albicans, 658
- Endothelioma of peritoneum, 966
- Enteralgia, 867  
 treatment, 869
- Enteritis, acute, 837  
 symptoms, 837  
 treatment, 838  
 as a cause of myocarditis, 229
- chronic, 839  
 diet in, 840  
 forms of, 839  
 symptoms, 839  
 treatment, 840
- chronic catarrhal, 839
- diarrhea and, 841
- in infants, 841  
 etiology, 841, 842  
 prophylaxis, 843  
 symptoms, 842  
 treatment, 843
- pseudomembranous, 839
- ulcerative, 839
- Enteroclysis, by Murphy method, 583, 940  
 continuous, 583  
 in peritonitis, 940, 944
- Enterocolitis, 841
- Enteroptosis, 862  
 diagnosis, 865  
 etiology, 863  
 Glénard's test, 865  
 symptoms, 864  
 treatment, 866  
 indications, 866
- Enterospasm, 868  
 treatment, 869
- Eosinophilia in asthma, 351
- Epidemic sore throat, 665
- Epinephrin in heart-block, 219  
 in heart diseases, 285
- Epistaxis, renal, 564
- Epithelial casts, 573

- Erythemia, 61  
 diet in, 62  
 symptoms, 61  
 synonym, 61  
 treatment, 62  
 X-ray, 62
- Esbach's albuminometer, 572
- Esbach test, 571
- Esophageal bougies, use of, 683.  
 See Bougie.  
 lavage, 815
- Esophagismus, 678  
 etiology, 678  
 treatment, 679
- Esophagitis, 812  
 acute, 674  
 etiology, 674  
 symptoms, 675  
 treatment, 675  
 diagnosis, 816  
 treatment, 816
- Esophagus, carcinoma of, 677  
 symptoms, 677  
 treatment, 677  
 diseases of, 674  
 diverticulum of, 679  
 diagnosis, 680  
 etiology, 679  
 prognosis, 680, 681  
 symptoms, 680  
 treatment, 680  
 foreign bodies in, 681  
 normal constrictions of, 684  
 stenosis of, 675  
 stricture of, 681  
 diagnosis, 682  
 etiology, 681  
 normal, 684  
 prognosis, 683  
 symptoms, 682  
 treatment, 683  
 ulcer of, 675  
 etiology, 675  
 symptoms, 676  
 treatment, 676
- Essential hematuria, 564, 565
- Exercises in chronic heart diseases, 292, 298  
 in pulmonary tuberculosis, 412, 414, 415, 416  
 Oertel's climbing, 297
- Exophthalmic goiter, 109  
 asthenic stage of, 110  
 erethic stage of, 109  
 etiology, 112, 114  
 ligation of thyroid vessels for, 117  
 myxedematous stage of, 110  
 symptoms, 112  
 pituitary extract in, 114
- Exophthalmic goiter, rest in, 114  
 results of thyroidectomy in, 117  
 sthenic stage of, 109  
 symptoms, 110  
 synonyms, 109  
 thymus involvement in, 112, 115  
 symptoms, 112  
 treatment, 118  
 tonsillectomy in, 114  
 transitional stage of, 109  
 symptoms, 112  
 treatment, 113  
 surgical, 116  
 X-ray, 115
- Expectoration in pulmonary tuberculosis, 377  
 disposal of, 404
- Exploratory laparotomy in gastric cancer, 735
- Extract, pituitary, 149. See Pituitary.
- Extrasystoles, 208  
 etiology, 208  
 treatment, 208
- Exudate *vs.* transudate, differentiation of pleural, 500
- Facial characteristics of pulmonary tuberculosis, 383
- Failure, causes of cardiac, 245  
 of cardiac compensation in nephritis, treatment of, 614
- Fatty casts, 573  
 degeneration of liver, 909. See Liver.  
 heart, 231  
 infiltration of liver, 910. See Liver.
- Febrile diarrhea, 841
- Fecal stasis, 559  
 diagnosis, 559
- Feeble breathing in tuberculosis, 395
- Feeding, duodenal, 714, 742
- Fetid stomatitis, 659. See Stomatitis, Ulcerative.
- Fever blisters, 649  
 etiology, 649  
 treatment, 649  
 hay-, 358. See Hay-fever.  
 in pulmonary tuberculosis, 379, 380, 456
- Fibers, elastic, in sputum, 379
- Fibrillation, auricular, 212  
 etiology, 212  
 in arteriosclerosis, 271  
 treatment, 273  
 prognosis, 214  
 symptoms, 212  
 treatment, 214

- Fibrinous bronchitis, 337. See  
     Bronchitis.  
     pericarditis, 222  
     pleuritis, acute, 502. See Pleu-  
         ritis.  
 Fibroid phthisis, 400, 401  
 Fibroma of adrenals, 91  
     of peritoneum, 963  
     of tongue, 671  
 Fibrous goiter, 123  
     tissue formation in tuberculosis,  
         371, 372  
 Fingers, clubbed, in tuberculosis,  
     384  
 Fissures of lips, 648  
     treatment, 649  
 Fistula-in-ano in pulmonary tuber-  
     culosis, 468  
 Floating kidney, 554. See also Kid-  
     ney, Movable.  
 Flutter, auricular, 211  
     prognosis, 212  
     symptoms, 211  
     treatment, 212  
 Follicular stomatitis, 656. See  
     Stomatitis Aphthous.  
 Foramen ovale, patent, 249.  
 Foreign bodies in esophagus, 681  
 Formol index, 733  
 Foul breath, 650  
     etiology, 650  
     treatment, 650  
 Fowler's position, 944  
 Fresh air treatment of pulmonary  
     tuberculosis, 409  
 Fröhlich's syndrome, 160  
 Function, tests for renal, 604  
 Functional hypoadrenia, 72  
     etiology, 73  
     prophylaxis, 74  
     symptoms, 72  
     treatment, 75  
  
 Gall-stone colic, 893  
     impaction, 892, 895  
 Gall-stones, 892  
     composition, 892  
     size, 892  
 Ganglioneuroma of adrenals, 91  
 Gangrene, pulmonary, 484  
     differentiated from abscess, 485  
     etiology, 484  
     pathology, 484  
     symptoms, 485  
     treatment, 485  
 Gangrenous stomatitis, 661. See  
     Stomatitis.  
 Gastrectasis, 818  
     acute, 818  
     continuous lavage in, 822  
 Gastrectasis, diagnosis, 821  
     from megalogastria, 818  
     from pyloric obstruction, 818  
     etiology, 819  
     forms of, 818, 819  
     objective findings in, 820  
     prognosis, 821  
     symptoms, 820  
     treatment, 821  
 Gastric abscess, 757  
     crises of cerebrospinal syphilis,  
         826  
     diagnosis, 829  
     prognosis, 830  
     spinal fluid in, 830  
     symptoms, 827  
     treatment, 831  
         between attacks, 835  
     lavage, 767, 769  
         in acute peritonitis, 942  
         in gastric carcinoma, 739  
 Gastritis, 749  
     acida, 762  
     acute catarrhal, 750  
         infectious, 755  
             etiology, 755  
             pathology, 756  
             prognosis, 756  
             symptoms, 756  
             treatment, 756  
             serum, 756  
         phlegmonous, 757  
             etiology, 757  
             mortality, 759  
             pathology, 758  
             symptoms, 758  
             treatment, 759  
         simple, 750  
             etiology, 750  
             prognosis, 750  
             treatment, 751  
                 of fever, 753  
                 of nausea, 753  
                 of pain, 753  
                 of vomiting, 752  
         suppurative, 757  
     alcoholic, 779  
         treatment, 779  
     anacida, 762  
         treatment, 778, 779  
     atrophic, 761  
         diagnosis, 761  
     chronic, 759  
         diet in, 764  
         drugs in, 776  
         etiology, 759  
         forms of, 762  
         lavage in, 767, 769  
         mineral waters in, 770  
         prognosis, 762



- Gastritis, treatment, 763  
 balneological, 770  
 dietetic, 764  
 direct, 764  
 electrical, 773  
 mechanical, 767  
 medicinal, 776  
 prophylactic, 763  
 croupous, 756  
 diagnosis, 749  
 diphtheritic, 756  
 hypertrophic glandular, 760  
 diagnosis, 760  
 membranous, 756  
 subacida, 762  
 treatment, 774, 778  
 toxic, 753  
 etiology, 753  
 pathology, 754  
 prognosis, 754  
 treatment, 754  
 types of, 750  
 Gastroenterostomy for gastric cancer, 738  
 Gastro-intestinal disturbances in leukemia, 41  
 in pulmonary tuberculosis, 381, 441  
 Gastroptosis, 862. See also Enteroptosis.  
 Gaucher's disease, 48, 49  
 type of splenomegaly, 48, 49  
 Geographical tongue, 669  
 treatment, 670  
 Germicidal action of thyroid secretion, 97  
 Gigantism, 151  
 symptoms, 152  
 treatment, 154  
 Gingivitis, 650  
 etiology, 650  
 treatment, 651  
 Glands, diseases of salivary, 672  
 inflammation of parotid, 672  
 Glénard's disease, 862  
 test, 865  
 Glomerulonephritis, 578, 579, 585  
 Glossitis, acute, 668  
 etiology, 668  
 prognosis, 668  
 symptoms, 668  
 treatment, 668  
 chronic, 669  
 etiology, 669  
 symptoms, 669  
 treatment, 671  
 Gluzinski test, 692, 733  
 Goiter, 118  
 colloid, 123  
 congenital, 120, 126  
 Goiter, treatment, 126  
 constrictive, 123  
 cystic, 123  
 etiology, 119  
 exophthalmic, 109. See Exophthalmic Goiter.  
 fibrous, 123  
 hemorrhagic, 123  
 hyperthyroid, 119, 122  
 treatment, 122  
 hypothyroid degenerative, 120, 123  
 etiology, 123  
 symptoms, 123  
 treatment, 124  
 varieties, 123  
 indications for operation in, 124  
 intrathoracic, 123  
 lingual, 123  
 malignant, 120, 125  
 treatment, 125  
 simple hypothyroid non-toxic, 119, 120  
 etiology, 120  
 treatment, 120  
 synonyms, 118  
 toxic, 119, 122  
 treatment, 122  
 types of, 119  
 Gonorrhea of mouth, 667  
 Grafting, thyroid, 105  
 Granular breathing in tuberculosis, 395  
 casts, 573  
 Grave's disease, 109  
 Green cancer, 43  
 sickness, 9  
 Grocco's triangle, 517  
 Gummata, of liver, 912  
 of lungs, 489, 491  
 of tongue, 670  
 Gymnastic exercises in heart diseases, 292  
 Haeser, coefficient of, 547  
 Hanot's cirrhosis of liver, 899  
 Hay-asthma, 351, 358. See also Hay-fever.  
 Hay-fever, 358  
 etiology, 358  
 prophylaxis, 360  
 treatment, 359  
 specific, 360  
 Heart action, normal, 242  
 rapid, 247  
 slow, 242, 243  
 alternation of, 219  
 diagnosis, 220  
 etiology, 219  
 prognosis, 220  
 treatment, 221

- Heart-block, 216, 243  
 etiology, 216  
 in arteriosclerosis, 271  
   treatment, 273  
 prognosis, 218  
 symptoms, 217  
 treatment, 218  
 dilatation of, 244, 246  
   cause, 245  
 disease in nephritis, 600  
 drugs, use of, 278  
 exercises in chronic diseases of, 292  
 failure, causes of, 245  
 fatty, 231  
 hidden focus of infection in disturbances of, 215  
 hypertrophy of, 244, 246, 254  
   in aneurysm, 262  
   in arteriosclerosis, 268  
   in nephritis, 602  
 irregularities of, 202  
 massage in diseases of, 296  
 muscle, functions of, 203  
 palpitation in tuberculosis, 382  
 physiology of, 203  
 pituitary extract in diseases of, 285  
 premature contractions of, 208, 243  
   etiology, 208  
   treatment, 208  
 rate of beat, 242  
 rheumatism of, 237, 240  
 senile, 256  
 shaggy, 222  
 stimulation of, through adrenals, 71  
 valves, rupture of, 254  
 valvular disease of, 242. See Valvular Disease.
- Heller's blood test, 567, 571  
 Hemangioma of tongue, 671  
 Hematemesis in anemia, 28  
   in cirrhosis of liver, 905  
 Hematoma of adrenals, 87  
   treatment, 88  
 Hematuria, 563  
   determining source of bleeding in, 564  
   diagnosis, 564  
   from hemoglobinuria, 564, 566  
   essential, 564  
   treatment, 565  
   etiology, 563  
   idiopathic, 564  
   treatment, 565  
   treatment, 565  
 Hemic murmurs, 6, 10  
 Hemoglobinuria, 565  
   Hemoglobinuria, diagnosis, 567  
   from hematuria, 564, 566  
   etiology, 566  
   paroxysmal, 566  
   symptoms, 566  
   treatment, 568  
 Hemopericardium, 222  
 Hemophilia, 56  
   coagulation time in, 57  
   differentiated from purpura, 58  
   etiology, 56, 57  
   hereditary transmission of, 57, 58  
   renal, 564  
   synonym, 57  
   treatment, 59  
   by transfusion, 59  
   serum, 59  
 Hemoptysis, causes of, 380, 451  
   treatment, 452, 453  
 Hemorrhages, adrenal, 85  
   cerebral, in nephritis, 616  
   in bronchiectasis, 342  
   in cirrhosis of liver, 905  
   in gastric ulcer, 692  
   treatment, 698, 721  
   in leukemia, 43  
   in pernicious anemia, 28  
   pancreatic, 916  
   pulmonary, causes of, 380, 381  
   renal, causes of, 563  
 Hemorrhagic diathesis, 56  
   peritonitis, 931  
   pleuritis, 526  
 Hemothorax, 528  
   treatment, 529  
 Henle, loop of, 545  
 Henoch's purpura, 54  
 Herpes labialis, 649  
   etiology, 649  
   treatment, 649  
 Herpetic stomatitis, 656. See Stomatitis, Aphthous.  
 Herzfehlerzellen, 475  
 Hippuric acid, 551  
   amount of, 551  
   identification of, 551  
   origin of, 551  
 His, bundle of, 203  
 Hoarseness in pulmonary tuberculosis, 380  
 Hodgkin's disease, 44  
   arsenic in, 46  
   blood picture in, 45  
   differential diagnosis, 45  
   prognosis, 45  
   salvarsan in, 46  
   symptoms, 44  
   synonyms, 44  
   tonsillectomy in, 47  
   treatment, 45

- Hodgkin's disease, treatment, serum,  
47  
surgical, 47  
tuberculin, 46  
vaccine, 46  
X-ray, 46
- Hoffman's sign, 132
- Home treatment of tuberculosis,  
407, 408
- Horseshoe kidney, 553
- Hospital treatment of pulmonary  
tuberculosis, 406, 407, 408
- Hot air bath, technic, 587
- Hot pack, technic, 586
- Hour-glass contraction of stomach,  
692
- Hum, venous, in anemia, 6  
in chlorosis, 10
- Hyaline casts, 572, 573
- Hydronephrosis, 625  
acquired, 625  
congenital, 625  
diagnosis, 626  
etiology, 625  
symptoms, 625  
treatment, 626
- Hydropericardium, 222
- Hydrotherapy in chlorosis, 13  
in pulmonary tuberculosis, 422,  
456
- Hydrothorax, 528  
etiology, 528  
symptoms, 528  
treatment, 528
- Hyperadrenia, 84  
etiology, 84  
treatment, 86
- Hyperemia of kidney, 575. See  
Kidney.
- Hypernephroma, 88  
of adrenals, 90  
of kidney, 89, 639  
diagnosed from calculus, 89  
symptoms, 88  
treatment, 89
- Hyperplasia of thymus, 140  
etiology, 140  
treatment, 143
- Hyperpituitaria, 149  
and diabetes insipidus, 150, 162  
etiology, 150  
following hypopituitaria, 150, 153,  
159, 160  
treatment, 155  
surgical, 155  
indications for, 155, 156
- Hypersecretion of salivary glands,  
672. See Ptyalism.  
of stomach in ulcer, 692  
treatment, 719
- Hypertension, 276, 277  
in nephritis, treatment of, 615  
treatment, 277
- Hyperthyroid goiter, 119, 122
- Hyperthyroidia, 109. See Exoph-  
thalmic Goiter.
- Hypertrophic cirrhosis of liver, 898.  
See Liver.
- emphysema, 362  
glandular gastritis, 760  
pulmonary osteoarthropathy, 342  
pyloric stenosis, 804
- Hypertrophy of heart, 244, 246, 254  
in aneurysm, 262  
in arteriosclerosis, 268  
in nephritis, 602
- Hyphomycetic stomatitis, 658. See  
Thrush.
- Hypoadrenalism, 72
- Hypoadrenia, 72  
chronic progressive, 80  
etiology, 80  
pathology, 80  
symptoms, 80, 81  
treatment, 81  
classes, 72  
functional, 72  
etiology, 73  
prophylaxis, 74  
symptoms, 72  
treatment, 75  
terminal, 77  
etiology, 77  
pathology, 77  
symptoms, 78  
treatment, 79
- Hypodermic administration of iron,  
12
- Hypoepinephry, 73
- Hypogastric neuralgia, 867  
treatment, 869
- Hypoparathyroidia, 129  
symptoms, 129  
treatment, 131
- Hypopituitaria, 160  
etiology, 162  
Lorain type of, 161  
secondary to hyperpituitaria, 150,  
153, 159, 160  
symptoms, 160  
treatment, 163
- Hypotension, 277
- Hypothyemia, 135  
etiology, 135
- Hypothyroid, degenerative goiter,  
120, 123, 124
- non-toxic goiter, 119, 120
- Hypothyroidia, 98  
and idiocy, 136  
etiology, 100

- Hypothyroidia, progressive, 102. See Myxedema.  
 symptoms, 99  
 treatment, 101
- Hypothyroidism, 98
- Idiocy and hypothyroidia, 136  
 and the thymus gland, 135, 136  
 Mongolian, 136  
 pathology, 137  
 symptoms, 136  
 treatment, 137
- Idiopathic hematuria, 564, 565
- Iliac abscess, 849  
 phlegmon, 849
- Immunity in tuberculosis, 373
- Impaction of gall stones, 892, 895
- Incomplete myxedema, 98
- Index, formul, 733  
 peptic, 733
- Indican, test for, 561
- Infantile myxedema, 103. See Cretinism.  
 splenic anemia, 50  
 arsenic in, 51  
 etiology, 50  
 iron in, 51  
 pathology, 51  
 symptoms, 50  
 synonym, 50  
 treatment, 51
- Infantilism, myxedematous, 106  
 treatment, 107
- Infections of mouth, 645  
 pulmonary, 463, 464, 472
- Infectious gastritis, acute, 755. See Gastritis.
- Inflammatory diarrhea, 841
- Inhalations in bronchiectasis, 344  
 in bronchitis, 330
- Ink polygraph, 203, 205
- Inoculation test for tubercle bacilli, 379
- Instruments, blood-pressure, 274
- Insufficiency, parathyroid, 129  
 symptoms, 129  
 treatment, 131  
 pineal, 172  
 pituitary, 160  
 testicular, 186, 187  
 thyroid, 98. See Hypothyroidia.
- Interlobular emphysema, 362
- Interstitial emphysema, 362
- Intestinal atony, 867  
 treatment, 869  
 neuroses, 867  
 treatment, 868  
 obstruction, 859  
 acute, 859  
 etiology, 859
- Intestinal atony, acute, symptoms, 860  
 treatment, 861  
 chronic, 859  
 etiology, 860  
 symptoms, 860  
 treatment, 862  
 complete, 859  
 etiology, 859, 860  
 incomplete, 859  
 mortality, 861  
 treatment, 861  
 types of, 859  
 parasites, anemia due to, 6  
 tuberculosis, 446, 871. See Tuberculosis.  
 in infants, 871  
 primary, 871  
 secondary, 872  
 symptoms, 871  
 treatment, 872
- Intestines, carcinoma of, 869  
 etiology, 869  
 symptoms, 870  
 treatment, 870  
 diminished sensibility of, 867  
 treatment, 869  
 diseases of, 837  
 neuralgia of, 867  
 treatment, 869  
 secretory disturbances of, 867  
 strangulation of, 859  
 etiology, 859  
 treatment, 861  
 syphilis of, 872  
 pathology, 872  
 sex incidence of, 873  
 treatment, 873  
 tuberculosis of, 446, 871. See Tuberculosis.
- Intrathoracic goiter, 123
- Intratracheal injections in bronchiectasis, 345
- Intussusception, 859  
 etiology, 859  
 treatment, 861
- Inunctions, oil, 451
- Iodides as cardiac drugs, 284
- Iodin, absorption of, by thyroid, 95  
 compound, Kendall's alpha, 95, 97
- Iodothyrim, 92, 94
- Iodothyroglobulin, 94
- Ipecac in pyorrhea, 654
- Iron, hypodermic administration, 12  
 in anemia, 7  
 in chlorosis, 11  
 in infantile splenic anemia, 51  
 in leukemia, 39  
 in purpura, 55  
 in tuberculosis, 459

- Irregularities, cardiac, 202  
Isthmus, Kronig's 392, 393
- Jaffe's test, 551
- Jaundice, 877  
  acute infectious, 881  
  avenues of infection in, 887  
  etiology, 881  
  history of, 882  
  mortality from, 881, 883  
  prevalence of, 881, 882, 886  
  among rats, 887  
  among troops, 886  
  seasonal, 884, 885  
  rats as carriers of, 882, 885, 886, 887  
  symptoms, 881, 883  
  transmission of, 884  
  treatment, 890
- albuminuria in, 569
- catarrhal, 877  
  diet in, 879  
  etiology, 877, 878  
  pathology, 878  
  prognosis, 879  
  symptoms, 878  
  treatment, 879  
  etiology, 877  
  trench, 883
- Jejunostomy, 738
- Joffroy's sign, 10
- Karrell diet in myocarditis, 234
- Kendall's alpha iodine compound, 95, 97
- Kidney, abscess of, 627  
  absence of, 552  
  adenoma of, 639  
  amyloid degeneration of, 618  
  etiology, 618  
  pathology, 618  
  symptoms, 618  
  treatment, 619  
  anatomy of, 542  
  anemia of, 574  
  etiology, 574  
  treatment, 575  
  anomalies of, 552  
  arteriosclerotic, 599  
  blood supply of, 544  
  carcinoma of, 639  
  circulatory disturbances of, 574  
  congenital cystic degeneration of, 639  
  etiology, 639  
  pathology, 639  
  diseases of, 541  
  displacements of, 553  
  acquired, 553  
  congenital, 553
- Kidney, floating, 554. See "Movable" Kidney, below.  
  histology of, 543  
  horseshoe, 553  
  hyperemia of, 575  
  etiology, 575  
  passive, 576  
  prognosis, 576  
  symptoms, 575  
  treatment, 576  
  hypernephroma of, 89, 639  
  large white, 591  
  malformation of, 552  
  movable, 553, 862  
  classification of, 553  
  diagnosis, 556, 557  
  etiology, 554, 864  
  indications for operation in, 561  
  symptoms, 555  
  treatment, 541, 557  
  nerve supply of, 544  
  palpable, 553  
  palpation of, technic, 556  
  physiology of, 545  
  ptosis of, 553  
  passive hyperemia of, 576  
  differentiated from nephritis, 577  
  etiology, 576  
  pathology, 576  
  symptoms, 577  
  treatment, 577
- pyogenic infections of, 626  
  etiology, 627  
  indications for operation in, 631  
  pathology, 627  
  prophylaxis, 629  
  symptoms, 628  
  treatment, 630
- sarcoma of, 639
- stones, 619. See also Nephrolithiasis.  
  composition, 619
- tuberculosis of, 633  
  diagnosis, 635  
  diet in, 638  
  pathology, 633  
  source of infection in, 633  
  symptoms, 634  
  treatment, 636  
  tuberculin, 638
- tumors, 639  
  symptoms, 639  
  treatment, 639  
  white, 591
- Klondike bed, 410
- Koch's bacillary emulsion, 431  
  old tuberculin, 431
- Kronig's isthmus, 392, 393



- Laboratory findings in gastric carcinoma, 734
- Laennec's cirrhosis of liver, 899.  
See Liver.
- Laparotomy, exploratory, in gastric cancer, 735
- Large-lunged emphysema, 362
- Large white kidney, 591
- Laryngitis, tuberculous, 467  
treatment, 467
- Launois's disease, 151
- Lavage, esophageal, 815  
gastric, 767, 769  
in acute peritonitis, 942  
in gastric carcinoma, 739  
continuous, 822
- Lenhartz diet, 703
- Leucocytic casts, 573
- Leucoplakia, 670  
etiology, 670  
symptoms, 670  
treatment, 671
- Leukemia, 29  
acute, 30  
arsenic in, 37  
atoxyl in, 38  
bacterial toxins in, 40  
benzol in, 35  
blood picture in, 31  
cardiac disturbances in, 42  
chronic, 30  
defibrinated blood in, 40  
diet in, 33  
etiology, 29  
gastro-intestinal disturbances in, 41  
iron in, 39  
lymphatic, 30  
myelogenous, 30  
salvarsan in, 36  
sodium cacodylate in, 39  
splenectomy in, 41  
thorium-X in, 37  
treatment, 32  
of dropsy, 42  
of hemorrhages, 43  
of special symptoms, 41  
tuberculin, 40  
X-ray, 33  
contraindications to, 34  
types of, 30  
varieties of, 30
- Ligation of thyroid vessels in exophthalmic goiter, 117
- Lightning pains in anemia, 29
- Lingual goiter, 123
- Lipoma of adrenals, 91  
of peritoneum, 963
- Lips, fissures of, 648  
treatment, 649
- Liver, abscess of, 905  
emetin in, 908  
etiology, 906  
sequelæ, 907  
symptoms, 906  
treatment, 907
- acute yellow atrophy of, 908  
etiology, 909  
pathology, 908  
symptoms, 909  
treatment, 909
- adenoma of, 913
- amyloid disease of, 911  
etiology, 911  
symptoms, 911  
treatment, 911
- anatomical relations of, 874
- angioma of, 913
- carcinoma of, 913
- cirrhosis of, 898  
atrophic, 898  
pathology, 899  
biliary, 898, 899  
etiology, 899, 900  
pathology, 899  
symptoms, 902  
diet in, 903  
etiology, 875, 898, 900  
Hanot's, 899  
hemorrhages in, 905  
hypertrophic, 898  
etiology, 900  
pathology, 899  
symptoms, 901
- Laennec's, 899
- portal, 898  
atrophic, 899  
forms of, 899  
hypertrophic, 899  
symptoms, 901  
of terminal stage, 905  
treatment, 902  
of hemorrhages, 905  
surgical, 904  
types of, 898
- cysts of, 914
- diseases of, 874
- fatty degeneration of, 909  
etiology, 909  
pathology, 909  
symptoms, 910  
treatment, 910
- fatty infiltration of, 910  
pathology, 910  
symptoms, 910  
treatment, 911
- physiology of, 874
- sarcoma of, 913
- sclerosis of, 898
- syphilis of, 912

- Liver, syphilis of, acquired, 912  
     congenital, 912  
     pathology, 912  
     symptoms, 912  
     treatment, 913  
     tuberculosis of, 913  
     tumors of, 913  
     symptoms, 913  
 Local emphysema, 362  
     peritonitis, chronic, 945, 946. See  
         Peritonitis, Chronic.  
 Loculated empyema, 522  
     diagnosis, 525. See also Em-  
         pyema.  
 Loop of Henle, 545  
 Lorain type of hypopituitarism, 161  
 Loss of weight in tuberculosis, 381  
 Ludwig's angina, 674  
 Lungs, bacterin treatment of dis-  
     eases of, 534  
     carcinoma of, 496  
     echinococcus disease of, 497  
     gummata of, 489, 491  
     sarcoma of, 496  
     tumors of, 495  
 Lymphadenoma, 44  
 Lymphatic leukemia, 30  
 Lymphemia, 31  
 Lymphocarcinoma and Hodgkin's  
     disease, 45  
 Lymphosarcoma and Hodgkin's dis-  
     ease, 45  
  
 Mackenzie's ink polygraph, 203, 205  
 Macroglossia, 672  
 Malformations of kidney, 552  
 Malignant endocarditis, 237  
     goiter, 120, 125  
 Malpighian pyramid, 543  
 Marie's disease, 151  
 Massage in chlorosis, 13  
     in heart diseases, 296  
 McBurney's point, 850  
 Mechanical purpura, 52  
     treatment of chronic gastritis, 767  
 Megalogastric, 818  
 Membrane, Cargile, 950, 951  
 Membranous gastritis, 756  
 Menstrual disorders, influence of  
     ovaries on, 179  
 Menstruation, vicarious, in phthisis,  
     381  
 Mercurial stomatitis, 662  
     symptoms, 662  
     treatment, 662  
 Metallic tinkle, 532  
 Metastasis in gastric carcinoma,  
     728  
     in gastric sarcoma, 746, 748  
 Miliary tuberculosis, 400  
  
 Milk and eggs in tuberculosis, 417,  
     418, 419  
 Mineral waters in chronic gastritis,  
     770  
 Mitral regurgitation, 250  
     frequency of, 250  
     physical signs of, 250  
     prognosis, 248, 250  
     with stenosis, 250  
     stenosis, 251  
         embolism in, 249  
         etiology, 247  
         physical signs of, 252  
         prognosis, 248  
         stages of, 252  
         with mitral regurgitation, 250  
         with tricuspid stenosis, 255  
 Moist râles in tuberculosis, 397  
 Mongolian idiocy, 136  
     pathology, 137  
     symptoms, 136  
     treatment, 137  
 Morbus maculosus, 54  
 Morphin in heart affections, 284  
 Mouth, diseases of, 643  
     as related to systemic diseases,  
         643  
     treatment of, 647  
     dry, 672  
     gonorrhea of, 667  
         treatment, 667  
     infections of, 645  
     sepsis of, 646  
         as a cause of cancer, 726  
         treatment, 695  
     syphilitic affections of, 666  
         treatment, 666  
 Movable kidney, 553. See Kidney.  
 Mucous colitis, 845. See Colitis.  
 Murexid test, 551  
 Murmurs, hemic, in anemia, 6  
     in chlorosis, 10  
 Murphy method of enteroclysis, 583,  
     940  
 Mycotic stomatitis, 658. See  
     Thrush.  
 Myeloma, 31  
 Myelogenous leukemia, 30  
 Myocarditis, 228  
     and valvular disease, 243  
     cane sugar in, 234  
     chronic, 230  
         symptoms, 230  
     diagnosis, 229, 230  
     drugs in, 235  
     elimination in, 232  
     etiology, 228  
     Karrell diet in, 234  
     prognosis, 230  
     rest in, 232

- Myocarditis, sequels to, 231  
 symptoms, 229  
 treatment, 231  
   indications for, 231  
   of symptoms, 235
- Myxedema, 102  
 etiology, 102, 103  
 incomplete, 98  
 infantile, 103. See Cretinism.  
 symptoms, 102  
 treatment, 103
- Myxedematous infantilism, 106  
 treatment, 107
- Nauheim baths, 286  
 administration technic, 287
- Nausea in acute gastritis, treatment of, 753
- Neoplasms, peritoneal, 963. See Peritoneal.  
 pulmonary, 495. See Pulmonary.  
 See also Tumors.
- Neosalvarsan in aneurysm, 264  
 in pernicious anemia, 21, 22
- Nephritic test diet, 605
- Nephritis, 578  
 acute parenchymatous, 578  
   complications, 582  
   diet in, 584  
   edema in, 581, 585  
   electric light bath in, 587  
   etiology, 578  
   dry cupping in, 589  
   glomerular type, 578, 579, 585  
   hot air bath in, 587  
   hot pack in, 586  
   pathology, 579  
   pericarditis in, 582  
   pilocarpin in, 588  
   dangers of, 588  
   prognosis, 582  
   prophylaxis, 582  
   purpura in, 582  
   symptoms, 580  
   treatment, 583  
     of edema, 585  
   tubular type, 578, 579, 580  
   uremia in, 582  
   urine in, 581  
   venesection in, 588  
 and arteriosclerosis, 268, 600  
 chronic interstitial, 599  
   association with heart disease, 600  
   cardiac hypertrophy in, 602  
   diet in, 612  
   dyspnea, toxic, in, 603  
   treatment, 616  
   etiology, 599  
   pathology, 600
- Nephritis, chronic interstitial, relation to arteriosclerosis, 268, 609  
 retinal changes in, 603  
 symptoms, 601  
 tests for renal function in, 604  
   creatinin in blood, 610  
   nephritic test diet, 605  
   phenolsulphonethalein, 604  
   urea in blood, 608  
   uric acid in blood, 606  
 treatment, 611  
   climatic, 612  
   of cerebral hemorrhage, 616  
   of dyspnea, 616  
   of failure of cardiac compensation, 614  
   of hypertension, 615  
   of uremia, 617  
 uremia in, 604  
 symptoms, 604  
 treatment, 617  
 urine in, 602  
 chronic parenchymatous, 589  
   anemia in, 592  
   diet in, 594  
   diuretics in, 597  
   edema in, 592  
   etiology, 590  
   paracentesis abdominis in, 598  
   pathology, 591  
   prognosis, 592  
   symptoms, 591  
   thoracentesis in, 597  
   treatment, 593  
   tuberculosis as a cause of, 590  
   urine in, 591  
 clinical classification of, 541, 578  
 complicating pulmonary tuberculosis, 464, 465  
 differentiated from passive hyperemia of kidney, 577  
 pathological types of, 578  
 suppurative, 626
- Nephrolithiasis, 619  
 etiology, 620  
 symptoms, 621  
 treatment, 624  
   of renal colic, 622
- Nephroptosis, 553, 862. See Kidney, Movable.
- Nervous diarrhea, 868  
 treatment, 869  
 symptoms in pulmonary tuberculosis, 383, 449
- Neuralgia, hypogastric, 867  
 treatment, 869  
 of intestines, 867  
 treatment, 869
- Neuroses of intestines, 867  
 treatment, 868

- Neurotic purpura, 52  
 Nitrites, action of, 283  
   dosage, 283  
   indications for use of, 283  
   in pulmonary tuberculosis, 453  
 Nitroglycerin, 283  
 Node, auriculoventricular, 203  
   sinoauricular, 203  
 Noma, 661. See Stomatitis, Gangrenous.  
 Non-exudative peritonitis, chronic  
   general, 952  
 Normal blood-pressure, 270, 276  
 Obstruction, intestinal, 859. See  
   Intestinal Obstruction.  
   pyloric, 801. See Pyloric Obstruction.  
 Oertel's climbing exercises, 297  
 Oil inunctions, 451  
 Old tuberculin, Koch's, 431  
 Oöphorectomy, effects of, 174, 178, 179  
   in osteomalacia, 175  
 Open-air schools, 440  
 Opium in pulmonary tuberculosis,  
   449, 453  
 Oral sepsis, 646  
   as a cause of cancer, 726  
   treatment, 695  
 Osteoarthropathy, hypertrophic pulmonary, 342  
 Osteomalacia, oöphorectomy in, 175  
 O. T., 431  
 Ovarian insufficiency, 177  
   symptoms, 177  
   treatment, 180  
   overactivity, 182  
   symptoms, 182  
   treatment, 183  
 Ovaries, active principles of, 175  
   and menstrual disorders, 179  
   disorders of, 173  
   effect of removal of, 174, 178, 179  
   functions of, 173, 174, 175  
   transplantation of, 181  
 Overactivity, pituitary, 149. See  
   Hyperpituitaria.  
   testicular, 190, 191  
   thyroid, 109. See Hyperthyroidia  
   and Exophthalmic Goiter.  
 Pacemaker, 203  
 Pack, technic of hot, 586  
 Pain in gastric cancer, 729  
   treatment of, 744  
 Pains, thoracic, in tuberculosis, 383  
 Palpable kidney, 553  
 Palpation of kidney, technic, 556  
 Pancarditis, 238  
 Pancreas, carcinoma of, 921  
   Pancreas, carcinoma of, symptoms, 921  
   treatment, 922  
   diseases of, 914  
   physiology of, 914  
   rupture of, 936  
 Pancreatic calculi, 919  
   etiology, 919  
   symptoms, 919  
   treatment, 919  
   cysts, 920  
   etiology, 920  
   symptoms, 920  
   treatment, 921  
   hemorrhage, 916  
   etiology, 916  
   symptoms, 916  
   treatment, 916  
 Pancreatitis, acute, 916  
   etiology, 916  
   symptoms, 917  
   treatment, 917  
   chronic, 918  
   etiology, 918  
   symptoms, 918  
   treatment, 919  
 Paracentesis abdominalis, technic,  
   598, 969  
   of pericardium, 227  
   thoracis, 514, 597  
   dangers, 521  
   indications, 516  
   technic, 517, 597  
 Paraganglioma of adrenals, 91  
 Paralysis agitans, parathyroids in,  
   132, 133  
 Parasites, anemia due to intestinal, 6  
 Parasitic form of thyroiditis, 108  
   stomatitis, 658. See Thrush.  
 Parathyroids, 93, 126  
   anatomy of, 126  
   and paralysis agitans, 132, 133  
   effect of removal of, 94, 127  
   functional disorders of, 132  
   histology of, 127  
   insufficiency of, 129  
   symptoms, 129  
   treatment, 131  
   organic disorders of, 132  
   physiology of, 127  
 Paratyphlitis, 849  
 Parietal peritoneum, 928  
 Parotid glands, inflammation of, 672  
   etiology, 673  
   treatment, 673  
 Parotitis, 672  
 Paroxysmal hemoglobinuria, 566  
   tachycardia, 209  
   diagnosis, 209  
   prognosis, 209

- Paroxysmal tachycardia, treatment, 210  
 Parry's disease, 109  
 Passive hyperemia of kidney, 576  
 Patent foramen ovale, 249  
 Pectoriloquy, 398  
 Peliosis rheumatica, 54  
 Pelvic peritonitis, 953. See Peritonitis.  
 Peptic index, 733  
 Percussion of chest, technic, 391  
 Periapical abscesses, 653  
 Pericardial adhesions, 222, 225  
   effusion, 222, 225  
 Pericarditis, 222, 223  
   dry, 222, 223, 224  
     etiology, 223  
     symptoms, 224  
   etiology, 223  
   fibrinous, 222  
   in nephritis, 582  
   plastic, 222  
   treatment, 226  
   with effusion, 222, 225  
     symptoms, 225  
     treatment, 227  
 Pericardium, adherent, 222, 225  
   affections of, 222  
   hemo-, 222  
   hydro-, 222  
   paracentesis of, 227  
   physiology of, 222  
 Perigastric abscess, 692  
 Perinephritic abscess, 632  
   etiology, 632  
   symptoms, 632  
   treatment, 632  
 Peritoneal neoplasms, 963  
   benign, 963  
     symptoms, 965  
     treatment, 966  
   malignant, 966  
     treatment, 966  
 Peritoneum, absorptive power of, 928  
   anatomy, 927  
   carcinoma of, 966  
   cysts of, 963  
   danger zones of, 929  
   diseases of, 927  
   endothelioma of, 966  
   fibromata of, 963  
   functions of, 930  
   lipomata of, 963  
   lymphatics of, 930  
   nerve supply of, 929  
   parietal, 928  
   sarcoma of, 966  
   secretory function of, 929  
   tumors of, 953  
 Peritoneum, visceral, 928  
 Peritonitis, acute general, 927  
   bacteria found in, 931  
   cause of death in, 935  
   due to colon bacilli, 933  
     to pneumococci, 931  
     to staphylococci, 933  
     to streptococci, 932  
   hemorrhagic type, 931  
   indications for drainage in, 939  
     technic, 939  
   indications for operation in, 937  
   leucocytosis in, 934  
   pathology, 930  
   purulent type, 931  
   serofibrinous type, 931  
   serous type, 931  
   sources of infection in, 935  
   symptoms, 932, 933  
   treatment, 937  
     enteroclysis, 940, 944  
     feeding, 943  
     gastric lavage, 942  
     position of patient, 944  
     stimulation, 943  
     surgical, 938  
     use of cathartics, 945  
   types, 931  
   appendicular, 959  
     pathology, 959  
     symptoms, 959  
     treatment, 960  
   chronic, 945  
     etiology, 945  
     general, 946  
       non-exudative, 952  
       symptoms, 948  
       treatment, 951  
     local, 945, 946  
       etiology, 946  
       pathology, 946  
       prophylaxis, 949, 950  
       symptoms, 946, 947  
       treatment, 949, 950  
     non-exudative general, 952  
       treatment, 952  
     pathology, 945  
     treatment, 949  
   non-exudative chronic general, 952  
     treatment, 952  
   pelvic, 953  
     differential diagnosis, 956  
     etiology, 953  
     pathology, 953  
     symptoms, 955  
     treatment, 957  
   subacute general, 935  
     symptoms, 935  
   tuberculous, 946



- Peritonitis, tuberculous, treatment, 951
- Perityphlitis, 849
- Pernicious anemia, 17  
arsenic in, 21, 22, 23  
blood transfusion in, 23  
defibrinated blood in, 24  
diet in, 21  
hemorrhages in, 28  
neosalvarsan in, 21, 22  
pathology, 17  
rest in, 21  
salvarsan in, 21, 22  
contraindications, 22  
sensory disturbances in, 29  
serums in, 25  
splenectomy in, 26  
contraindications, 26  
mortality from, 26  
results of, 26  
symptoms, 20  
treatment, 20  
of special symptoms, 27  
X-ray, 27  
venesection in, 24
- Phenolsulphonethalein test, 604
- Phlebograms, 205
- Phlegmon, iliac, 849
- Phlegmonous gastritis, 757. See Gastritis.
- Phosphates in urine, 552  
amount of, 552  
origin of, 552  
tests for, 552
- Phosphatic calculi, 620
- Phthisical chest, 384
- Phthisis, 371. See Tuberculosis, Pulmonary.  
fibroid, 400, 401  
florida, 400  
syphilitic, 492
- Pilocarpin in nephritis, 588  
dangers of use, 588
- Pineal gland, anatomy of, 168  
diseases of, 168  
function of, 169  
histology of, 168  
insufficiency, 172  
sand, 168  
tumors, 171  
symptoms, 171  
treatment, 172
- Pine-stem artery, 271
- Pituitary body, anatomy of, 145  
connection of, with adrenals, 71  
diseases of, 145  
effect of removal of, 146  
histology of, 145  
physiology of, 146  
extract, effect of, 149
- Pituitary extract in exophthalmic goiter, 114  
in heart diseases, 285  
insufficiency, 160. See Hypopituitaria.  
overactivity, 149. See Hyperpituitaria.  
of anterior lobe, 150  
of posterior lobe, 150  
tumors, 151  
symptoms, 151  
treatment, 155
- Pityriasis versicolor in pulmonary tuberculosis, 384
- Plastic pericarditis, 222
- Pleura, bacterin treatment of diseases of, 534  
carcinoma of, 526  
sarcoma of, 526
- Pleural effusion, 499, 509, 510. See Pleural Fluids.  
in pulmonary tuberculosis, 460  
exudate, 500  
fluids, 499, 510  
bacteriology of, 502  
cytology of, 501  
transudate, 500  
tumors, 526  
diagnosis, 527  
symptoms, 526  
treatment, 527
- Pleurisy. See Pleuritis.
- Pleuritis, acute fibrinous, 502  
etiology, 502  
pathology, 503  
physical signs, 504  
symptoms, 504  
strapping of chest in, 505  
treatment, 505  
operative, 505  
hemorrhagic, 526  
etiology, 526  
in pulmonary tuberculosis, 502, 503, 507
- purulent, 521. See Empyema.
- serofibrinous, 509  
aspiration of fluid, 516  
diagnosis, 511  
etiology, 509  
exploratory puncture in, 514  
pathology, 510  
physical signs, 511  
symptoms, 511  
thoracentesis, 516  
dangers of, 521  
indications, 516  
technic, 517, 520  
treatment, 515
- Pneumococci as cause of peritonitis, 931

- Pneumoconiosis, 486  
 etiology, 486  
 pathology, 486  
 prophylaxis, 488  
 symptoms, 487  
 treatment, 488
- Pneumonia, tuberculous, 400  
 white, 489
- Pneumopericardium, 222
- Pneumothorax, 529  
 etiology, 530  
 in bronchiectasis, 348  
 in pulmonary tuberculosis, 462, 533  
 pathology, 529, 530  
 physical signs, 531  
 symptoms, 531  
 treatment, 532
- Point, McBurney's, 850
- Polycythemia, 61
- Polygraph, ink, 203, 205
- Porches, sleeping, 409
- Portal cirrhosis of liver, 898, 899.  
 See Liver.
- Position, Fowler's, 944
- Postoperative tetany, 129  
 symptoms, 129  
 treatment, 131
- Postural treatment of bronchiectasis, 346
- Pregnancy, complicating tuberculosis, 471  
 tubal, differentiated from pelvic peritonitis, 956
- Premature contractions of heart, 208, 243  
 auricular, 208  
 etiology, 208  
 in arteriosclerosis, 270  
 treatment, 208
- Prevention of tuberculosis, 402  
 by public education, 405
- Progressive hypoadrenia, 72. See Hypoadrenia.  
 hypothyroidia, 102. See Myxedema.
- Protein sensitization tests in asthma, 352, 353
- Proteinuria, Bence-Jones, 570
- Pseudoangina, 255
- Pseudoleukemia, 44
- Pseudomembranous enteritis, 839
- Ptoxis of kidney, 553
- Ptyalism, 672  
 etiology, 672  
 treatment, 672
- Pulmonary abscess, 478. See Abscess.  
 actinomycosis, 493. See Actinomycosis.
- Pulmonary aspergillosis, 499  
 blastomycosis, 499  
 carcinoma, 496  
 congestion, 474. See Congestion.  
 distomatosis, 499  
 edema, 476. See Edema.  
 acute suffocative, 476  
 gangrene, 484. See Gangrene.  
 hemorrhages, causes of, 381  
 infections, chronic non-tuberculous, 472  
 etiology, 473  
 in children, 473  
 pathology, 473  
 physical signs, 473  
 symptoms, 472  
 treatment, 473  
 neoplasms, 495  
 benign, 495  
 diagnosis, 496  
 malignant, 495  
 symptoms, 496  
 treatment, 497  
 sarcoma, 496  
 stenosis, 248, 250, 255  
 streptothricosis, 494  
 syphilis, 489  
 tuberculosis, 371  
 tumors, 495
- Pulse, rapid, 242  
 rate in tuberculosis, 382  
 slow, 242, 243
- Pulsus alternans, 219
- Purpura, 52  
 arthritic, 52  
 cachectic, 52  
 differentiated from hemophilia, 58  
 etiology, 52  
 hemorrhagica, 54  
 Henoch's, 54  
 in nephritis, 578  
 mechanical, 52  
 neurotic, 52  
 peliosis rheumatica, 54  
 rheumatica, 52  
 simplex, 53  
 etiology, 53  
 treatment, 54
- Purulent peritonitis, 931  
 pleuritis, 521. See Empyema.
- Putrid sore throat, 659. See Stomatitis, Ulcerative.
- Pyelitis, 626
- Pyelonephritis, 626  
 suppurative, 626
- Pyloric obstruction, 801  
 differentiated from gastrectasis, 818  
 from ulcer, 692  
 etiology, 801

- Pyloric obstruction, prognosis, 802  
   symptoms, 801  
   treatment, 802  
 Pyloric stenosis, congenital, 804  
   classification, 804  
   diagnosis, 807  
     differential, 808  
   etiology, 804  
   mortality, 808  
   objective findings, 807  
   pathology, 805  
   prognosis, 808  
   symptoms, 806, 807  
   treatment, 808  
 Pylorospasm, 796  
   etiology, 796  
   prognosis, 798  
   symptoms, 797  
   treatment, 799  
   types, 797  
 Pyogenic infections of kidney, 626.  
   See Kidney.  
 Pyonephrosis, 627  
 Pyopneumothorax, 533  
   differentiated from subphrenic ab-  
   scess, 962  
 Pyorrhea alveolaris, 651  
   diagnosis, 653  
   emetin hydrochlorid in, 654  
   etiology, 651  
   ipecac in, 654  
   symptoms, 653  
   treatment, 654  
  
 Radium treatment of gastric cancer,  
   745  
 Râles in pulmonary tuberculosis,  
   396, 397  
 Rapid heart action, 242  
 Rats as carriers of *spirochæta*  
   *icterohæmorrhagicæ*, 882,  
   885, 886, 887  
 Reaction, Wolff-Junghan, 733  
 Rectal feeding in gastric cancer, 742  
 Regurgitation, aortic, 253  
   etiology, 253  
   physical signs in, 253  
   prognosis, 248  
   sudden death in, 248  
   mitral, 250  
     frequency of, 250  
     physical signs, 250  
     prognosis, 250  
     with stenosis, 250  
   tricuspid, 254  
     etiology, 254  
     prognosis, 248  
     symptoms, 254  
 Renal calculus diagnosed from hy-  
   pernephroma, 89  
  
 Renal colic, 621  
   etiology, 621  
   treatment, 622  
   epistaxis, 564  
   function tests, 604  
   hemophilia, 564  
   hemorrhages, causes of, 563  
 Resection of stomach, 736  
 Resistance exercises in heart dis-  
   ease, 292, 298  
 Resonance, vocal, in pulmonary  
   tuberculosis, 398  
 Rest in myocarditis, 232  
   in pulmonary tuberculosis, 412  
 Rests, adrenal, 68, 88  
 Retention of urine, differentiated  
   from suppression, 562  
 Retinal changes in nephritis, 603  
 Rhagades of commissures, 648  
 Rheumatic purpura, 52  
 Rheumatism of heart, 237, 240  
 Rupture of heart valves, 254  
   of pancreas, 936  
  
 Salivary calculi, 673  
   glands, diseases of, 672  
   hypersecretion of, 672. See  
   Ptyalism.  
 Salts, urinary, 552  
 Salvarsan as a cardiac drug, 283  
   in aneurysm, 263  
   in Hodgkin's disease, 47  
   in leukemia, 36  
   in pernicious anemia, 21, 22  
 Sanatorium treatment of tubercu-  
   losis, 406  
   advantages, 407  
   disadvantages, 407  
 Sand, pineal, 168  
 Sarcoma, and Hodgkin's disease, 45  
   of kidney, 639  
   of liver, 913  
   of lungs, 496  
   of peritoneum, 966  
   of pleura, 526  
   of stomach, 746. See Stomach.  
   of tongue, 671  
 Schiff's test, 550  
 Schönlein's disease, 54  
 Schools, open-air, 440  
 Sclerosis of liver, 898  
   of tubercles, 371, 372  
 Secretory disturbances of intestines,  
   867  
   function of peritoneum, 929  
 Semicretins, 104  
 Senile emphysema, 362, 369  
   pathology, 369  
   symptoms, 370  
   treatment, 370

- Senile heart, 256  
 Sensibility, diminished intestinal, 867  
     treatment, 869  
 Sensitization tests, protein, 352, 353  
 Sepsis, oral, 646  
     as a cause of cancer, 726  
     treatment, 659  
 Sergeant's white line, 80  
 Serofibrinous peritonitis, 931  
     pleuritis, 509. See Pleuritis.  
 Serous peritonitis, 931  
 Serums, in gastric cancer, 746  
     in hemophilia, 59  
     in Hodgkin's disease, 47  
     in pernicious anemia, 25  
 Shaggy heart, 222  
 Sibilant râles in tuberculosis, 397  
 Sickness, green, 9  
 Siderosis, 486  
 Sign, Bacelli's, 514  
     Chvostek's, 132  
     Hoffman's, 132  
     Joffroy's, 10  
 Silicosis, 486  
 Silver nitrate in ulcer of stomach, 700  
 Sinoauricular node, 203  
 Sinus arrhythmias, 207  
 Sippy's treatment of gastric ulcer, 701  
 Skodaic resonance, 393  
 Sleeping porches, 409  
 Slow heart action, 242, 243  
 Small-lunged emphysema, 362  
 Smithies' diet, 716  
 Smoking in pulmonary tuberculosis, 447  
 Sodium bicarbonate in gastric ulcer, 701  
     cacodylate in anemia, 7  
     in leukemia, 39  
     chloride in urine, 552  
     amount of, 552  
     origin of, 552  
 Solids, urinary, 547  
 Sonorous râles in tuberculosis, 397  
 Sore throat, epidemic, 665  
     putrid, 659. See Stomatitis, Ulcerative.  
 Sores, cold, 649  
 Sources of infection in tuberculosis, 402  
 Specific treatment of pulmonary tuberculosis, 428  
 Spermine, 185, 186  
 Sphygmograms, 205  
 Spirals, Curshmann's, 351  
 Spirochaeta icterohaemorrhagicae, 881  
     detection of, 888, 889, 890  
 Spirochaeta icterohaemorrhagicae, rats  
     as carriers of, 882, 885, 886, 887  
     recovery of, from urine, 889  
 Splanchnoptosis, 862. See also Enteroptosis.  
 Splash, succussion, 532  
 Splenectomy in leukemia, 41  
     in pernicious anemia, 26  
     contraindications, 26  
     mortality, 26  
     results, 26  
 Splenic anemia, 47  
     infantile, 50  
     arsenic in, 51  
     etiology, 50  
     iron in, 51  
     pathology, 51  
     symptoms, 50  
     synonyms, 50  
     treatment, 51  
     pathology, 48  
     symptoms, 49  
     treatment, 49  
     X-ray, 50  
     varieties of, 48  
     von Jaksch's, 48  
 Splenomegaly, 47, 48  
 Sponging in pulmonary tuberculosis, 422, 423, 457  
 Sporadic cretinism, 104  
 Sputum, albumin reaction of, 379  
     -cups, 405  
     elastic fibers in, 379  
     in tuberculosis, 378  
     tubercle bacilli in, 378  
 Staining tubercle bacilli, 378  
 Staphylococci as cause of peritonitis, 933  
 Stasis, diagnosis of fecal, 559  
 Status lymphaticus, 140  
     diagnosis, 140  
     symptoms, 140  
     treatment, 143  
     thymicolymphaticus, 140  
     symptoms, 142  
 Stenosis, aortic, 254  
     congenital pyloric, 804. See Pyloric Stenosis.  
     mitral, 251  
     embolism in, 249  
     etiology, 247  
     physical signs, 252  
     prognosis, 248  
     stages of, 252  
     with mitral regurgitation, 250  
     with tricuspid stenosis, 255  
     of esophagus, 675  
     pulmonary, 248, 250, 255  
     tricuspid, 255  
     prognosis, 248

- Stenosis, tricuspid, with mitral stenosis, 255
- Stereoscopic examination in pulmonary tuberculosis, 398
- Stokes-Adams syndrome, 217
- Stomach, acute dilatation of, 818.  
See Gastrectasis.
- carcinoma of, 724  
age incidence of, 725  
diagnosis, 728  
laboratory, 731, 733, 734  
diet in, 740  
duodenal feeding in, 742  
etiology, 725  
exploratory laparotomy in, 735  
frequency of, 724  
gastric analysis in, 731  
lavage in, 739  
gastroenterostomy for, 737  
gastrostomy for, 738  
histology of, 727  
jejunostomy for, 738  
laboratory findings in, 734  
metastasis in, 728  
pain in, 729  
prognosis, 727  
conditions influencing, 727, 734  
rectal feeding in, 742  
resection of stomach for, 736  
site of growth, 728  
symptoms, 729  
of inoperability, 730  
of obstruction, 730  
treatment, 735  
hygienic, 739  
medical, 738, 742  
of anemia, 743  
of constipation, 745  
of pain, 744  
radium, 745  
serum, 746  
surgical, 735  
X-ray, 745  
types of, 728  
of operation for, 735  
vomiting in, 729, 730
- catarrh of, 759  
diseases of, 684  
resection of, 736
- sarcoma of, 746  
duration of, 746  
etiology, 747  
frequency of, 746  
histology of, 747  
incidence of, 747  
location of, 747  
metastasis in, 746, 748  
pathology, 747  
physical signs, 748
- Stomach, sarcoma of, symptoms, 748  
treatment, 749
- syphilis of, 780  
diagnosis, 784  
X-ray, 783  
forms of, 781  
frequency of, 780  
gastric analysis in, 783  
histology, 782  
prognosis, 785  
symptoms, 782, 783  
treatment, 785
- tuberculosis of, 789  
avenues of infection in, 790  
diagnosis, 791  
diet in, 793  
forms of, 789  
frequency of, 789  
pathology, 791  
prognosis, 792  
symptoms, 791  
treatment, 792  
tuberculin, 795  
ulcer of, 684. See Ulcer.
- Stomatitis, aphthous, 656  
etiology, 656  
symptoms, 656  
treatment, 657
- catarrhal, 655  
etiology, 655  
treatment, 656
- fetid, 659. See Stomatitis, Ulcerative.
- follicular, 656. See Stomatitis, Aphthous.
- gangrenous, 661  
etiology, 661  
symptoms, 661  
treatment, 662
- herpetic. See Stomatitis, Aphthous.
- hyphomycetica, 658. See Thrush.
- mercurial, 662  
symptoms, 662  
treatment, 662
- mycotic, 658. See Thrush.
- parasitic, 658. See Thrush.
- ulcerative, 659  
etiology, 659  
symptoms, 659  
treatment, 660
- vesicular. See Stomatitis, Aphthous.
- Stones, calcium oxalate, 619  
kidney, 619. See Nephrolithiasis.  
phosphatic, 620  
ureteral, 621  
uric acid, 619
- Strangulation of bowel, 859  
etiology, 859



- Strangulation of bowel, treatment, 861
- Strapping the chest, technic, 428, 505
- Streptococci as cause of peritonitis, 932
- Streptothricosis, pulmonary, 494  
   etiology, 494  
   pathology, 495  
   symptoms, 495  
   treatment, 495
- Stricture of esophagus, 681. See Esophagus.
- Strophanthin, 283
- Strophanthus, 282  
   indications for, 282
- Struma, 118
- Strychnin as a cardiac drug, 286
- Subacid gastritis, 762  
   treatment, 774, 778
- Subacute bronchitis, 326. See Bronchitis.  
   general peritonitis, 935
- Sublingual ulcer, 671
- Succussion splash, 532
- Suffocative pulmonary edema, 476.  
   See Edema, Pulmonary.
- Sugar in myocarditis, 234
- Summer diarrhea, 841
- Suppression of urine, 561. See Anuria.
- Suppurative gastritis, acute, 757  
   nephritis, 626
- Swedish movements in heart disease, 296
- Syndrome, Fröhlich's, 160  
   Stokes-Adams, 217
- Syphilis, cerebrospinal, gastric crises in, 826  
   complicating pulmonary tuberculosis, 469  
   mouth lesions in, 666  
   of intestines, 872, 873  
   of liver, 912. See Liver.  
   of stomach, 780. See Stomach.  
   pulmonary, 489  
     as gummata, 489, 491  
     pathology, 489, 490  
     symptoms, 491  
     treatment, 492
- Syphilitic affections of mouth, 666  
   phthisis, 492
- Systemic diseases as related to diseases of the mouth, 643
- Systoles, premature, 208, 243  
   in arteriosclerosis, 270
- Tabloid, Bland, 236
- Tachycardia, paroxysmal, 209  
   diagnosis, 209
- Tachycardia, paroxysmal, prognosis, 209  
   treatment, 210
- "Taking the cure," 412, 413
- Teeth, abscess of, in heart disturbances, 215  
   and tuberculosis, 646  
   infections, as cause of disease, 644
- Tent, window, 410
- Terminal hypoadrenia, 77  
   etiology, 77  
   pathology, 77  
   symptoms, 78  
   treatment, 79
- Test, Binet-Simon, 139  
   coin, 532  
   complement fixation, in tuberculosis, 375
- Edestin, 733
- Esbach, 571  
   for albuminuria, 570, 571  
   for creatinin in blood, 610  
   for urea in blood, 608  
   for uric acid in blood, 606
- Glenard's, 865
- Gluzinski, 692, 733
- Heller's blood, 567, 571
- indican, 561
- inoculation, for tubercle bacilli, 379
- Jaffe's, 551
- murexid, 550
- nephritic test diet, 605
- phenolsulphonaphthalein, 604
- protein sensitization, 352, 353
- renal function, 604
- Schiff's, 550
- Von Pirquet's, 374
- Weyl's, 551
- Testicles, disorders of, 184  
   internal secretion of, 184  
   transplantation of, 188
- Testicular insufficiency, 186  
   etiology, 187  
   symptoms, 186  
   treatment, 187  
   overactivity, 190  
   symptoms, 190  
   treatment, 191
- Tests, protein sensitization, 352, 353
- Tetany, 130, 132, 133  
   etiology, 132  
   postoperative, 129  
   symptoms, 129  
   treatment, 131  
   symptoms, 132  
   treatment, 133
- Thoracentesis, 514, 597  
   dangers, 521  
   indications, 516

- Thoracentesis, technic, 517, 597  
 Thoracic aneurysm, 259, 261  
 Thorium-X, 37  
 Throat, epidemic sore, 665  
   etiology, 665  
   treatment, 665  
 Thrush, 658  
   etiology, 658  
   symptoms, 658  
   treatment, 658  
 Thymectomy, 144  
   effect of, 134  
 Thymic asthma, 140, 142  
   treatment, 143  
   death, 140, 142  
   etiology, 142  
 Thymus gland, and idiocy, 135, 136  
   diseases of, 133  
   effect of removal, 134  
   function of, 135  
   histology of, 133  
   hyperplasia of, 140  
     etiology, 140  
     treatment, 143  
     X-ray, 143  
   in backward children, 138  
   in exophthalmic goiter, 112, 115  
   insufficiency of, 135. See Hypo-  
     thymia.  
 Thyroid, absorption of iodine, 95  
   gland, administration of dried, 101  
     anatomy of, 92  
     connection with adrenals, 71  
     diseases of, 92  
     nervous stimulation of, 95  
     physiology of, 92-98  
     result of excision of, 93  
     in exophthalmic goiter, 117  
   grafting, 105  
   insufficiency, 98. See Hypothy-  
     roidia.  
   overactivity, 109. See Hyperthy-  
     roidia and Exophthalmic  
     Goiter.  
   secretion, excessive, 94  
     germicidal action of, 97  
     influence of, on metabolism, 95  
 Thyroidase, 98  
 Thyroidectomy, results of, 93  
 Thyroiditis, 107  
   acute, 107  
     complications of, 107  
     etiology, 107  
     symptoms, 107  
     treatment, 108  
   chronic, 108  
     etiology, 108  
     treatment, 109  
   parasitic form, 108  
 Tinkle, metallic, 532  
 Tongue, carcinoma of, 671  
   enlargement of, 672. See Macro-  
     glossia.  
   diseases of, 668-672  
   geographical, 669  
     treatment, 670  
   gumma of, 670  
     treatment, 670  
   inflammation of, 668, 669. See  
     Glossitis.  
   tuberculosis of, 670  
     treatment, 670  
   tumors of, 671  
     treatment, 671  
   ulcer of, 671  
   white, 670. See Leucoplakia.  
 Tonsillectomy, in gastric ulcer, 696  
   in Hodgkin's disease, 47  
 Tonsils as source of systemic dis-  
   eases, 645  
 Toxic dyspnea in nephritis, 603  
   treatment, 616  
   gastritis, 753. See Gastritis.  
   goiter, 119, 122  
 T. R., 431  
 Transfusion of blood in pernicious  
   anemia, 23  
 Transplantation of ovaries, 181  
   of testicles, 188  
 Transudate *vs.* exudate, differentia-  
   tion of pleural, 500  
 Trench jaundice, 883  
 Triangle, Grocco's, 513  
 Tricuspid regurgitation, 254  
   etiology, 254  
   prognosis, 248  
   symptoms, 254  
   stenosis, 255  
     prognosis, 248  
     with mitral stenosis, 255  
 Tubal pregnancy differentiated from  
   pelvic peritonitis, 956  
 Tubercle bacilli, 378  
   inoculation test for, 379  
   staining of, 378  
 Tubercles, 371  
   calcification of, 371, 372  
   caseation of, 371, 372  
   sclerosis of, 371, 372  
 Tuberculin, 430  
   dosage, 432  
   in gastric tuberculosis, 795  
   in Hodgkin's disease, 46  
   in kidney tuberculosis, 638  
   in leukemia, 40  
   in phthisis, 430  
   contraindication to, 433  
   dosage, 432  
 Tuberculosis, bovine, 402, 403  
   intestinal, 446, 871

Tuberculosis, intestinal, frequency, 446  
 in infants, 871  
   primary, 871  
   secondary, 872  
   symptoms, 871  
   treatment, 872  
   symptoms, 446  
   treatment, 446  
 of kidney, 633. See Kidney.  
 of liver, 913  
 of stomach, 789. See Stomach.  
 of tongue, 670  
 pulmonary, 371  
   acute general miliary, 400  
   alcohol in, 429  
     objections to, 429, 430  
   ammonium chloride in, 449  
   anemia in, 382, 459  
     treatment, 459  
   anorexia in, 442  
   arsenic in, 459  
   as a cause of nephritis, 590  
   associated diseases, 468  
   artificial pneumothorax in, 423  
     contraindications, 424  
     dangers, 424  
     for hemoptysis, 455  
     indications, 424  
     results, 425  
     technic, 427  
   auscultation, 394  
   bathing, 422, 423, 456, 457  
   blistering in, 461  
   blood-pressure, 382  
   breath sounds, 394  
     types, 395  
   bronchitis in, 447  
   bubbling râles in, 397  
   calcification in, 371, 372  
   cardiac disease complicating,  
     468, 469  
     palpitation in, 382  
   caseation in, 371, 372  
   cavity formation in, 372, 397  
   chest, diminished expansion,  
     385, 386  
     inspection, 383, 384  
     mensuration, 388  
     pain, 449, 450  
     strapping, 428, 505  
   chronic ulcerative, 400, 401  
   class method of instruction, 435  
   climate in treating, 408  
   clubbed fingers, 384  
   cold bath, 422  
   cold sponges, 422, 423, 457  
   complement fixation test for  
     diagnosing, 375  
   complications, 460  
   constipation, 444

Tuberculosis, pulmonary, constipation, treatment, 446  
 cough, 377, 446  
   treatment, 446  
 creosote in, 448  
   indications, 448  
   method of administering, 448  
 curved finger nails in, 384  
 "danger zone," 397  
 determination of degree of  
   activity, 401  
   of improvement, 401  
 diabetes complicating, 470  
   treatment, 470  
 diagnosis, 383  
 diarrhea, 445  
   etiology, 445  
   treatment, 445  
 diet, 416  
   calories needed, 417  
   in children, 422  
   milk and egg, 417  
 dispensary treatment, 433  
   cases suitable, 434  
   functions, 441  
 eggs, method of taking, 418,  
   419  
 exercise, 412  
   contraindications, 414  
   deep breathing, 416  
   walking, 414  
   working, 415  
 expectoration, 377  
   disposal of, 404  
 facial characteristics, 383  
 fever, 379, 456  
   following exercise, 380  
   menstrual, 380  
   premenstrual, 380  
   treatment, 456  
 fistula-in-ano complicating, 468  
   significance, 468  
   treatment, 468  
 fibrous tissue formation, 371,  
   372  
 fresh air, 409  
 gastro-intestinal disturbances,  
   441  
 hemoptysis, 380, 451  
   causes, 451  
   treatment, 452, 453  
 hemorrhages, 380, 381  
 hoarseness, 380  
 home treatment, 407, 408  
   visitation, 437  
 hospital treatment, 406, 407, 408  
 hydrotherapy, 422, 456  
 immunity, 373  
 inspection, 383

- Tuberculosis, pulmonary, intestinal  
 tuberculosis complicating, 446  
   iron in, 459  
   Kronig's isthmus, 392, 393  
   location of first foci in, 371  
   loss of appetite in, 442  
     of weight, 381  
   method of dissemination, 404  
   miliary, 400  
   milk and eggs, 417, 418, 419  
   mixed infections complicating,  
     463  
     treatment, 464  
   mode of onset, 377  
   moist râles, 397  
   nephritis complicating, 464  
     treatment, 465  
   nitrites in, 453  
   oil inunctions, 451  
   opium in, 449, 453  
   pain in, 449  
     treatment, 450  
   palpation, 383  
   pathology, 371  
   patient's history, 376  
   pectoriloquy, 398  
   percussion, 390  
     technic, 391  
   physical signs, 383  
   pityriasis versicolor, 384  
   pleural effusion in, 460  
     treatment, 460  
   pleuritis, 502, 503, 507  
   pneumothorax complicating,  
     462, 533  
     cause, 462  
     treatment, 462  
   pregnancy complicating, 471  
     treatment, 471  
   prevention, 402  
     public education in, 405  
   pulse rate, 382  
   râles, 396  
     method of eliciting, 397  
     significance, 397  
   rest, 412  
   sanatorium treatment, 406  
     advantages, 407  
     disadvantages, 407  
   sibilant râles, 397  
   skodaic resonance, 393  
   smoking, 447  
   sonorous râles, 397  
   sources of infection, 402  
   sponging, 422, 423, 457  
   sputum, 378  
   stereoscopic examination, 398  
   symptoms, 377  
     cardiovascular, 382  
     gastro-intestinal, 381, 441
- Tuberculosis, pulmonary, symptoms,  
   nervous, 383, 449  
   syphilis associated with, 469  
     treatment, 469  
   "taking the cure," 412, 413  
   teeth and, 646  
   treatment, 399, 406  
     climatic, 408  
     dispensary, 433  
     fresh air, 409  
     general, 406  
     home, 407, 408  
     medicinal, 428  
     of special symptoms, 441  
     prophylactic, 402  
     specific, 428  
     tuberculin, 430  
   tuberculin treatment, 430  
     contraindication, 433  
     dosage, 432  
   tuberculous laryngitis compli-  
     cating, 467  
     treatment, 467  
   types, 400  
   vaccines in, 464  
   vicarious menstruation, 381  
   vocal resonance, 398  
   vomiting, 442  
     causes, 442, 443  
     treatment, 443  
   von Pirquet's test, 374  
   X-ray, 398  
     interpretation, 399  
     value, 398, 399
- Tuberculous adenitis differentiated  
   from Hodgkin's disease, 45  
   infection without disease, 374  
   laryngitis, 467  
     treatment, 467  
   peritonitis, 946  
     treatment, 951  
   pleurisy, 460  
     treatment, 460  
   pneumonia, 400  
   resistance, 372  
   ulcer of stomach and duodenum,  
     789
- Tubular breathing, 396  
 Tubulonephritis, 578, 579, 580  
 Tufnel's treatment of aneurysm, 265  
 Tumors, of adrenals, 90  
   of kidney, 639  
   of liver, 913  
   of pleura, 526  
   of tongue, 671  
   peritoneal, 963. See Peritoneal  
     Neoplasms.  
   pineal, 171  
   pituitary, 151  
   pulmonary, 495

- Ulcer of esophagus, 675  
 etiology, 675  
 symptoms, 676  
 treatment, 676  
 of stomach and duodenum, 684  
 acute dilatation of stomach in, 721  
 age incidence of, 687  
 breathing exercises in, 696  
 carcinoma following, 688, 691, 723  
 care of body in, 696  
 of mouth, 695  
 complications of, 688, 691  
 treatment, 718  
 diagnosis, 692  
 diet in, 703  
 daily schedule, 704-709  
 essentials of, 703  
 in follow-up treatment, 712  
 Lenhartz, 703  
 Smithie's, for emaciation, 716  
 von Leube, 703  
 drugs in, 700  
 duodenal feeding in, 714  
 objections to, 717  
 technic, 714  
 duration of, 688  
 Einhorn's duodenal feeding in, 714  
 objections to, 717  
 technic, 714  
 etiology, 684  
 frequency of, 686  
 general hygiene in treatment of, 695  
 hemorrhage from, 692  
 treatment, 698, 721  
 hour-glass contraction in, 692  
 hypersecretion in, 692  
 treatment, 719  
 local applications of heat and cold in, 697  
 location of ulcers in, 687  
 mortality, 688  
 number of ulcers in, 687  
 nutritive enemata in, 699  
 perigastric abscess from, 692  
 prognosis, 687  
 pyloric obstruction from, 692  
 results of operation for, 689  
 sex incidence of, 687  
 size of ulcers in, 687  
 tonsillectomy in, 696  
 treatment, 693, 700  
 alkali, 701, 702  
 daily schedule in, 705-710  
 essentials of, 693  
 follow-up, 710  
 of complications, 718
- Ulcer of stomach and duodenum,  
 treatment of hemorrhage,  
 698, 721  
 of hemorrhoids, 699  
 of hypersecretion, 719  
 of pain, 718  
 of perforation, 723  
 of relapsing cases, 714  
 of special symptoms, 718  
 of thirst, 698  
 of vomiting, 720  
 Sippy's, 701  
 tuberculous, 789  
 of tongue, 671  
 sublingual, 671  
 Ulcerative colitis, 848  
 enteritis, 839  
 stomatitis, 659. See Stomatitis.  
 Urea, 547  
 amount of, 547  
 estimation of, 549  
 formation of, 548  
 in blood, estimation of, 608  
 Uremia, 582, 604  
 treatment, 617  
 Ureteral stones, 621  
 Uric acid, 550  
 in blood, estimation of, 606  
 origin of, 550  
 stones, 619  
 tests for, 550  
 Urinary phosphates, 552  
 amount of, 552  
 origin of, 552  
 tests for, 552  
 salts, 552  
 solids, 547  
 estimation of, 547  
 Urine, anomalies of secretion of, 561  
 albuminuria, 569  
 anuria, 561  
 casts, 572  
 chyluria, 568  
 cylindroids, 574  
 hematuria, 563  
 hemoglobinuria, 565  
 characteristics of normal, 546  
 excretion of, 545  
 in acute parenchymatous nephritis, 581  
 in chronic interstitial nephritis, 602  
 in chronic parenchymatous nephritis, 591  
 retention of, differentiated from suppression, 562  
 suppression of, 561. See Anuria.  
 Vaccines in acute infectious gastritis, 756



- Vaccines in asthmatics, 355  
 in bronchiectasis, 345  
 in chronic bronchitis, 336  
 in chronic non-tuberculous pulmonary infections, 474  
 in endocarditis, 241  
 in Hodgkin's disease, 47  
 in pulmonary tuberculosis, 464  
 in treating diseases of lungs, bronchi, and pleura, 534
- Valves, rupture of heart, 254
- Valvular disease of heart, 242  
 and muscular disease, 243  
 congenital, 249  
 clinical pathology, 242  
 effects of, 243  
 general treatment, 246  
 pathologic physiology, 247  
 pathology, 243, 244, 245  
 prognosis, 248, 249  
 conditions influencing, 243  
 pulmonary, 255
- Venesection in aneurysm, 265  
 in pernicious anemia, 24  
 technic, 588
- Venous hum, in anemia, 6  
 in chlorosis, 10
- Vesicular emphysema, diffuse, 361, 362  
 etiology, 362  
 pathology, 363  
 symptoms, 364  
 treatment, 367  
 stomatitis, 656. See Stomatitis, Aphthous.
- Vicarious menstruation in pulmonary tuberculosis, 381
- Vincent's angina, 659. See Stomatitis, Ulcerative.
- Visceral crises, 826  
 peritoneum, 928
- Visceroptosis, 862. See also Enteroptosis.
- Vocal resonance in pulmonary tuberculosis, 398
- Volvulus, 859
- Vomiting in acute gastritis, treatment of, 752  
 in gastric carcinoma, treatment of, 729, 730
- Vomiting in pulmonary tuberculosis, 442, 443  
 in ulcer of stomach and duodenum, 720  
 von Jaksch's splenic anemia, 48  
 von Leube's diet, 703  
 von Pirquet's test, 374
- Walking exercise in pulmonary tuberculosis, 414
- Waxy casts, 574, 575
- Wenzel's classification of cretins, 104
- Weyl's test, 551
- White kidney, 591  
 line, Sergent's, 80  
 pneumonia, 489  
 tongue, 670. See Leucoplakia.
- Window tent, 410
- Wiring aneurysms, 266
- Woillez's disease, 474
- Wolff-Junghan reaction, 733
- Working exercise in pulmonary tuberculosis, 415
- Xerostoma, 672  
 etiology, 672  
 treatment, 672
- X-ray diagnosis, of bronchiectasis, 343  
 of pulmonary tuberculosis, 398, 399  
 of syphilis of stomach, 783  
 in erythemia, 62  
 in exophthalmic goiter, 115  
 in gastric carcinoma, 745  
 in Hodgkin's disease, 46  
 in leukemia, 33  
 in pernicious anemia, 27  
 in splenic anemia, 50  
 in thymic hyperplasia, 143
- Yellow atrophy of liver, acute, 908.  
 See Liver.
- Zone, danger, in pulmonary tuberculosis, 397  
 of peritoneum, 929













